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The Journal of
Bone and Joint Surgery

EDITORIALS AND ANNOTATIONS

SYME'S AMPUTATION

There is no general agreement about the merits or otherwise of Syme's amputation, although it is 112 years since it was first described. The subject still remains a useful one for any discussion group. An opportune article by the authoritative writer, R. I. Harris, appears in this issue, and there can be few who will disagree with his views. It seems that Syme's own modification of the original operation is the best of many. In this he removed the projecting malleoli, whereas his original operation was merely a disarticulation. In this country the modification of this amputation introduced by Elmslie in 1921 is now more frequently carried out, with removal of the tibial articular surface and three-quarters of an inch above it, and, of course, both malleoli. It is noted that Harris recommends leaving the articular surface and encourages new bone formation in this region, to form the bulbous end stumps which Syme attempted to achieve. The advantage of this shape of end stump is that it lessens the " piston-like " action inside the prosthesis, which is more commonly seen with the conical shaped stump resulting from a high level of amputation. If this action is severe it necessitates the wearing of a long prosthesis with weight bearing on the tibial condyles and the thigh.

Syme's amputation has had some hard criticism from English (as distinct from Scottish) surgeons, for in 1942 Langdale-Kelham and Perkins believed that the stump would not last more than eight years and that it never looked anything but artificial. They concluded by expressing the hope that the modified Syme's would soon be as dead as the original Syme's—and presumably, also, as Syme himself.

Harris, a great protagonist, says, on the other hand, that the great advantage is that it is the most useful of all amputations of the leg because " a more comfortable stump is provided, more seemly and useful for support and progressive motion."

A considerable advantage of this type of amputation is, of course, that the wearer may go about the house without crutches or other mechanical aid, and so it is particularly useful for old men at night. Indeed, one sardonic surgeon said it had its urological advantages in that it made micturition easier, but he added that that was hardly an indication for a Syme's amputation.

Canadian experience is very similar to Scottish, and most Scottish surgeons strongly favour that method in just those cases in which Harris says it is indicated and principally because it permits full weight bearing on the end of the stump and usually lasts the patient's life-time instead of the eight years suggested. Shelswell took a cross-section of sixty-six patients with a Syme's amputation whose stump gave them no trouble, and found that fifty-seven of these had no trouble after twenty years, the remainder being of shorter duration.

The reasons for differences of opinion about the operation are stated by Harris, and it appears that the principal one lies in the operation itself and in the after-treatment. Various imperfections of the end-result and the methods of their avoidance are described. Most of the imperfections can be avoided by a careful technique, and if this were generally practised there would be fewer complaints about this excellent operation.

Harris reminds us of the specialised form of elastic adipose tissue developed between the calcaneum and the plantar aponeurosis which is resistant to pressure. There are here

pockets of fat enclosed by dense septa of fibrous tissue. These fibrous tissue strands are in the form of the letter U, with the open end of the U pointing towards the calcaneum. If this concept is true it is obvious why the dissection of the heel flap should be close to the calcaneum, because if these little loculi are opened, as will happen if the dissection is through the subcutaneous layer, the fat content is extruded and an important weight-bearing mechanism rendered useless.

The plane of transection of the tibia should be so placed that the minimum of bone is removed and the largest possible cross-section of the tibia remains. This leaves a large area for the support of the heel flap and minimum localised areas of pressure.

Another imperfection that is commonly overlooked is the misplaced heel flap. So often after the operation the patient is sent out of the theatre to have the bandaging and the dressing completed. A little too much pull inwards or outwards produces—and permanently—a flap which is not exactly beneath the centre of the cut lower end of the tibia. Harris secures this correct position by strips of adhesive plaster. An equally good method—possibly even better in its certainty—is to use the minimum of dressing and then apply plaster-of-Paris.

In a recent review by Shelswell (1954) of 305 Syme's amputations in 302 patients it is interesting that the main causes of failure, requiring re-amputation and occurring in about 25 per cent of all cases, were sepsis, as an early complication, and vascular insufficiency as a later complication. Both of these should be avoidable and in any case suggest an error of technique or of indication—it may be both.

Syme's amputation, even when the operation is well done and when the errors of detail in after-care to which reference has been made have been avoided, is rarely advisable in women. The cosmetic difficulties of the provision of an elegant prosthesis have not yet been solved. It is, however, an excellent conservative amputation and is ideally suited for the manual worker. As has been shown, meticulous attention to detail, however apparently trivial, is essential if the operation is to give lasting satisfaction. WALTER MERCER.

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FRACTURES OF THE LATERAL CONDYLE OF THE TIBIA

Orthopaedic surgeons, with their weakness for the picturesque and the transatlantic, received the colloquialism " bumper fracture " in a ready and fond embrace. This happy gesture now seems to have sprung from an illusion: Mr Graham Apley, who writes on page 699, cannot incriminate motor cars (automobiles) in more than five of sixty cases of fracture of the lateral condyle of the tibia. Does the Briton perambulate more carefully, or does he more rarely survive an unequal encounter? Has the motorist become more careful or more lethal? Whatever the explanation, we are grateful to Mr Apley for an interesting corrective.

To the novice, the radiograph of a broken lateral tibial condyle, with gross involvement of a major weight-bearing joint, looks rather forbidding. Yet perhaps the most striking thing about such injuries is how well nearly all of them do if only given a chance. They are almost without the spectre of serious vascular calamity that haunts some fractures of the neighbourhood. The fracture, by its very presence, suggests—usually correctly—that the

ligaments are intact: in forcible abduction of the knee either bone yields or ligaments, but seldom both: consequently there is rarely need to immobilise for the sake of the ligaments. The break is in cancellous bone, which promises ready union though slow consolidation. Unless weight is borne, the fracture is stable. Consequently, as in some other fractures through cancellous bone, the key to treatment is early active movement, but prolonged relief from weight bearing. The days when limbs with these fractures were commonly encased in plaster and kept in it must surely be long past, just as the cuirass has been discarded for the uncomplicated fracture of the spine.

To this easy picture there are important exceptions, notably unacceptable knock-knee. Many devices have been used to elevate and mould the condyle, or to close a vertical split kept open by indriven meniscus or bony debris. Even if reduction succeeds, maintenance may be difficult; commonly it implies one of two evils, either plaster immobilisation or else recumbency with splinting or traction or both.

Against this background we may consider Mr Apley's paper. He describes first a mixed bag of a dozen patients treated by plaster fixation—three by open operation too; then, in reaction against this restrictive management, he describes a method of early mobilisation of the knee with recumbency and with traction through a tibial transfixion pin. This method he has used routinely: it is, he says, " without operation."

The innovation—if innovation it is—may well have much to offer in the exceptional case of special difficulty; but its indiscriminate adoption would be regrettable for the reasons that follow. In most patients good results are obtained by early active movements and prolonged relief from weight bearing by simpler, safer, cheaper and less irksome methods. Transfixion of bone " an inch or two beyond the fracture " is an operation, and is not without risk; at this point the pin may enter the fracture haematoma, and this usually communicates with the joint; one day a knee will be infected. Prolonged recumbency is at the best tedious and expensive, and at the worst fatal. If, then, this method seems to offer advantages, let it be reserved for the cases that do not promise a good result from simpler, safer measures.

Forty-one patients, out of forty-eight treated by this method, have been examined—twenty-seven of them after five years. Mr Apley has made the instructive and important observation that any clinical change after a year was for the better, and that some patients were without recognisable osteoarthritis at the end of fifteen years. Would an equal number of patients treated by orthodox methods and surviving this long have done as well? We do not know. If we try to compare Mr Apley's results with those of Mr Gerald Slee in the Journal of a year ago, we find that the two papers differ in the type of fracture, in the ages of the patients, in the duration of observation and in the methods of assessment. All we can say is that both series present very good results. H. JACKSON BURROWS.

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SYME'S AMPUTATION

The Technical Details Essential for Success

R. I. HARRIS, TORONTO, CANADA

There is considerable divergence of opinion about the merits of Syme's amputation. In some countries it is highly regarded; in some it is neglected; in others it even is roundly condemned. In Canada it is thought to be the best of all amputations of the lower extremity above the foot and has been used with consistent success since before the 1914–1918 war to the present day. Experience in Great Britain seems to have been different, for there is an influential and experienced body of opinion which condemns it and states that almost every Syme's amputation of the 1914–1918 war has had to be converted to a below-knee stump because the Syme's stump was unsatisfactory in various respects (Ministry of Pensions publication 1939). In the Amputation Centre in Nijmegen in the Netherlands Syme's amputation is not performed. In the United States before the recent war Syme's amputation was not highly regarded, but the International Conference on Amputations held in Ottawa and Toronto in March 1944 led to renewed interest in Syme's amputation in the United States and several papers have since appeared to record the satisfaction of American orthopaedic surgeons with this special amputation.

Such striking differences of opinion must be due to some fundamental difference in the technique of the amputation as performed in different countries. It is the purpose of this paper to set forth the technical details which we have found to be essential factors for success.

HISTORICAL REVIEW

It is interesting and informative to revert to Syme's original record of this historic landmark in amputation surgery (Fig. 1). His first publication relating to it was published in 1843, twenty-two years before his son-in-law, Joseph Lister, first embarked upon his experiments in antiseptic surgery and three years before Morton first used ether at the Massachusetts General Hospital, and four years before Simpson first recorded the use of chloroform.

Syme's amputation, therefore, was developed in the period of pre-anaesthetic and pre-antiseptic surgery and his concern was to achieve a simple and safe means of removing an infected or damaged foot. His first report in February 1843 in the *London and Edinburgh Monthly Journal of Medical Sciences* was entitled " Amputation at the Ankle Joint." In this he records the successful outcome of his treatment, one year previously, of a case of suppurative disease of the tarsus (probably tuberculosis) by disarticulation of the foot at the ankle and removal of the malleoli flush with the lower articular surface of the tibia. The heel flap was deliberately preserved and was used to cover the end of the tibia but without suture of the incision. The wound healed gradually in spite of an abscess which required drainage. The man left hospital three months after his operation with " the wounds soundly healed and any degree of pressure can be born (*sic*) by the stump which has a round end well suited for the adaption of a boot or an artificial foot and is strongly protected from external injury by its thick integuments."

Syme's interest in this disarticulation-amputation grew from his enthusiasm for Chopart's disarticulation which he was the first to employ in Great Britain. He had learned that this was a valuable procedure which permitted the removal of a crushed and infected forefoot with a minimum of risk to the patient. By its use the dangerous amputation through the leg

was rendered unnecessary and the patient was left with a support " not less useful than that which is afforded by the whole tarsus." Such success led naturally to consideration of disarticulation at the ankle joint when the calcaneum and talus were involved in disease.

By 1846 Syme had published five articles on this new amputation. In the last of these he was able to state: "I have operated upon more nearly two than one dozen of cases with perfect success." The operative technique had been modified and perfected. The lower articular surface of the tibia together with the malleoli were removed with a single sawcut, and precise details for fashioning and separating the heel flap were given.

Syme stated that the advantages of the operation were, " 1st, that the risk of life will be smaller; 2d, that a more comfortable stump will be afforded; 3d, that the limb will be more

FIG. 1

James Syme (1799–1870), Professor of Clinical Surgery, University of Edinburgh. This is Holl's engraving from George Richmond's drawing of him " in the prime of life." Probably this was his appearance at age forty-four when he published his first paper, " Amputation at the Ankle Joint." At his death he was memorialised thus by his pupil and friend, John Brown, " *Verax; Capax; Perspicax; Sagax; Efficax et Tenax.*"

seemly and useful for progressive motion." Reading between the lines there can be no doubt that in Syme's mind the importance of the first advantage transcended the others. This is confirmed by his modest boast of having operated upon nearly two dozen cases with perfect success at a time when injuries and infections of the foot could only be mastered by the most radical measures. The common treatment was amputation through the leg at the site of election (a hand's breadth below the knee) though the mortality from this amputation was between 25 to 50 per cent from infection in the widely opened tissue spaces and the open medullary cavities of the tibia and fibula (Godlee 1924).

Since Syme's day the risk of infection in surgery has been greatly reduced, first by the development of antiseptic surgery by his son-in-law, Joseph Lister, later by the perfection of aseptic surgery and finally by the introduction of bacteriostatic and antibiotic agents.

To-day the great advantage of Syme's amputation no longer is " that the risk of life will be smaller." It remains the most useful of all amputations of the leg because " a more comfortable stump is provided, more seemly and useful for support and progressive motion." To Syme must be given the credit for bringing to the attention of the surgical world the value of end-bearing in amputations of the lower extremity even though, in the surgical era in which he worked, the merit of end-bearing was secondary in importance to the saving of life.

To-day the functional value of end-bearing amputations of the lower extremity is so great that every effort should be made to use them when possible. Since we are more fortunately situated than James Syme in that we do not have to concern ourselves so greatly

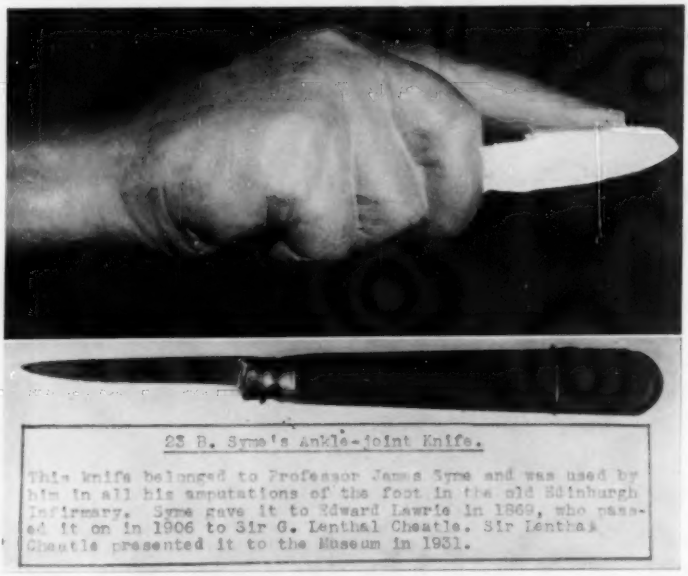
23 B. Syme's Ankle-joint Knife.
This knife belonged to Professor James Syme and was used by him in all his amputations of the foot in the old Edinburgh Infirmary. Syme gave it to Edward Lawrie in 1869, who passed it on in 1906 to Sir G. Lenthal Cheatle. Sir Lenthal Cheatle presented it to the Museum in 1931.

FIG. 2

Below—The " stout, sharp amputation knife " which Syme used to free the calcaneum from the heel flap. *Above*—Plaster model of Syme's right hand grasping the knife. Museum, Royal College of Surgeons of England.

with infection, we can concentrate our attention upon the development of the technique which will give the best possible end-bearing stump.

FUNDAMENTAL PRINCIPLES OF END-BEARING AMPUTATIONS

The important functions of the lower extremity are weight bearing and propulsion. Amputation stumps of the lower limb must be designed to suit these functions. The more perfectly they bear the weight of the body and transmit the forces of locomotion the more competently their prosthetic appliances will be used. For purposes of weight bearing nothing equals a stump which can bear weight upon its end. Propulsion is best performed by the stump which preserves the greatest length of limb with normally functioning muscles and joints above the level of the amputation. Syme's amputation, being end-bearing and of nearly normal length, offers the best possibilities of good function in amputations of the lower extremity.

To provide an end-bearing stump in the lower extremity certain requisites are essential. 1) The bone must be divided where its cross-section area is as great as possible in order to provide a broad area of support. 2) The whole of the cut surface of the bone must be capable of bearing weight. This can be achieved by a strong meshwork of cancellous bone across

the whole area, or in the case of the ankle joint by the retention of the subarticular cortical bone at the lower end of the tibia. The tubular cross-section of the shaft of the tibia at higher levels is unsuited to weight bearing. 3) The skin and subcutaneous tissue covering the end of the stump must be appropriate for weight bearing. Only two levels in the lower extremity meet these requirements. They are the lower end of the femur with a covering of prepatellar skin, and the expanded lower ends of the tibia and fibula covered by the heel pad.

It is interesting to learn that Syme attempted to devise an amputation at the level of

FIG. 3
Technique of Syme's amputation. Skin incisions carried down to bone and entry of the ankle joint.

the knee joint embodying the principles which proved so successful at the ankle joint. He reported his first two cases in 1845, two years after his first publication, "Amputation at the Ankle Joint." Both patients seem to have been suffering from tuberculosis of the knee joint. In both, the femur was transected through the condyles just above the articular surface, which was carious. The end of the stump was covered with a long posterior flap of skin derived from the calf. Both wounds healed without serious complication though they took some time to do so.

It is evident from Syme's presentation of these two cases that he was chiefly concerned with devising a safer operation than amputation through the mid-shaft of the femur and

believed that section of the bone where it was cancellous involved less risk from sepsis. He took no pains to cover the end of the stump with weight-bearing skin, evidently believing that the achievement of a healed stump without sepsis and without serious risk to the patient's life was all that was necessary to ensure good function and even weight bearing.

Twenty-one years later, in 1866, he wrote again about transcondylar amputation of the femur. His interest had been renewed by Carden's (1864) report of his method of amputating at this level using an anterior flap after removing the patella. Syme warmly commended Carden's

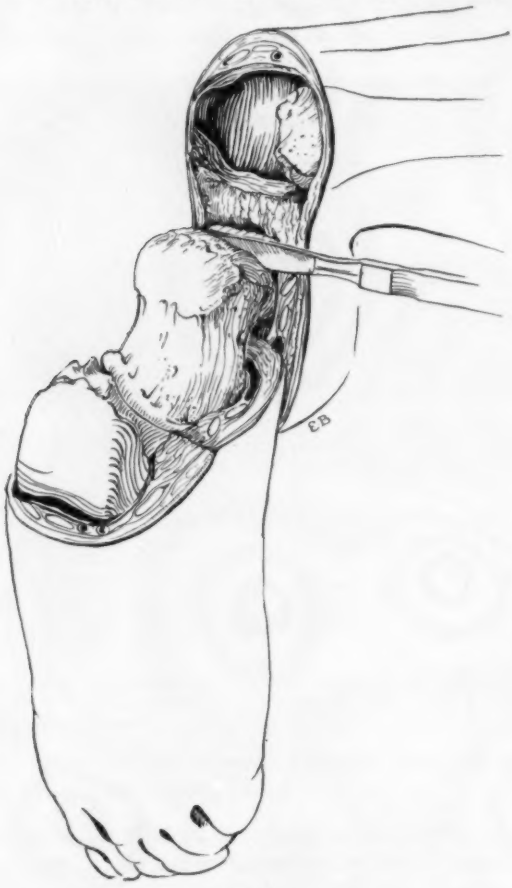

FIG. 4

Technique of Syme's amputation, continued. The talus has been dislocated from the ankle. The calcaneum has been nearly completely separated from the heel flap by subperiosteal dissection. The tendo calcaneus is about to be divided at its insertion.

amputation, which could be performed with little risk to the patient from sepsis with the additional advantage that " the stump proved eminently serviceable, since the skin over the bone, instead of becoming thinner, acquired additional thickness, so that the patients could rest upon it just as they do after amputation at the ankle." In this publication Syme acknowledged that his earlier attempt to perfect the technique of transcondylar amputation through the femur had failed and had fallen into disuse because the skin flap derived from the calf of the leg " proved very inconvenient."

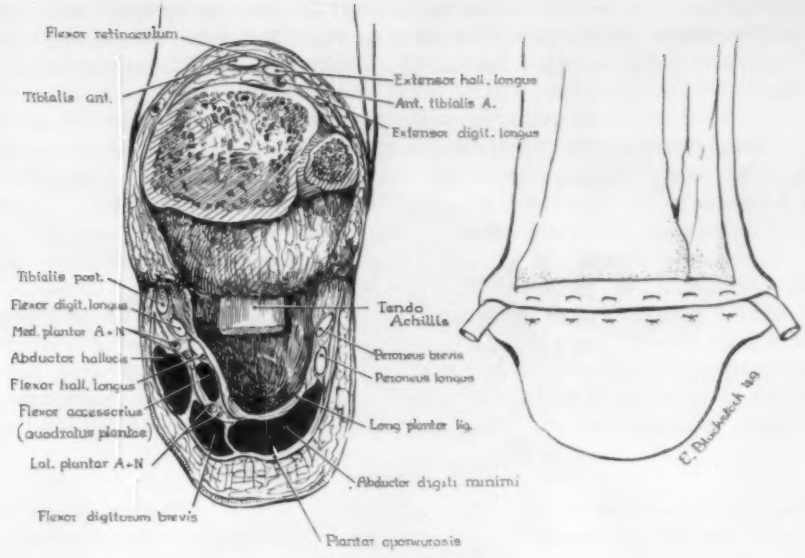

FIG. 5

Technique of Syme's amputation, continued. *Left*—The anatomy of the field of operation after the tarsus has been removed from the heel flap. *Right*—Closure of the wound with drainage.

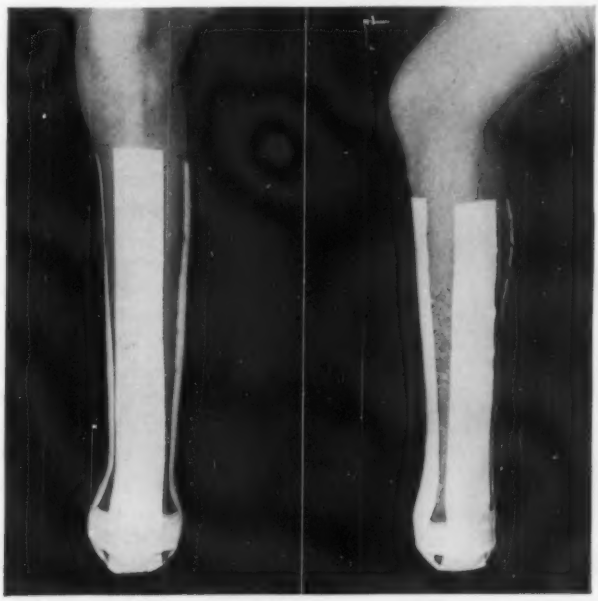

FIG. 6

Technique of Syme's amputation, continued. The method of strapping the heel flap to the leg to ensure that its position is exactly correct and will remain so.

Syme, therefore, nearly achieved success in devising an end-bearing stump at the transcondylar level. He failed because his attention was focused upon the avoidance of sepsis, and because he did not appreciate the importance of covering the end of the stump with weight-bearing skin, though in the case of the ankle joint he seems to have been aware of its value.

ADVANTAGES OF SYME'S AMPUTATION

The incomparable merit of Syme's amputation is that it permits full weight bearing on the end of the stump. Canadian experience indicates that this quality is retained until the end of the patient's life. The stump is nearly as long as a normal leg. This, added to the quality of end-bearing, gives the patient a stump which approaches the function of a normal foot. The patient can stand and walk upon it without limitations and can undertake the heaviest kind of work. He can even walk upon it without a prosthesis. It is by far the most useful and most serviceable of all amputations of the leg and should be used whenever possible. It is much more valuable than a below-knee amputation. Few below-knee amputees can walk on their prosthesis all day without damaging the stump, because weight is borne, not upon the end but upon the side of the stump, where there is no specialised weight-bearing skin and subcutaneous tissue and where the thrust is not end-bearing but is oblique, and is a shearing force.

STRUCTURE OF THE HEEL PAD

Many factors combine to make a Syme's amputation a good end-bearing stump, but the detail most essential for success is the preservation of the weight-bearing function of the heel flap. This function derives partly from the thickened skin but chiefly from the specialised structure of the elastic adipose tissue interposed between the skin on the one hand and the calcaneum and plantar aponeurosis on the other. Kuhns (1949) reviewed our knowledge of elastic adipose tissue and brought to our attention the detailed studies of Tietze (1921) and Blechschmidt (1933). Wherever pressure or weight bearing is applied to localised points on the body (heels, fingertips, thenar and hypothenar eminences, ischial tuberosities and prepatellar fat pads) a specialised form of elastic adipose tissue is developed which resists pressure. This quality is obtained by the presence of dense septa of elastic fibrous tissue enclosing spaces filled with fat. Each loculus is separate from its neighbour and the fat lobules within it are isolated from the surrounding loculi. In the heel pad the fibrous septa extend from the dermis below and are closed above and posteriorly by the inferior surface of the calcaneum and anteriorly by the plantar aponeurosis. They enclose flask-shaped spaces filled with fat lobules. Each space is reinforced by oblique and spirally arranged bands. These compartments, bounded by sheets of elastic fibrous tissue and filled with semi-fluid fat, act as hydraulic buffers. Under pressure they change their form but not their content. When pressure is released they resume their normal shape (Figs. 8 to 13).

FIG. 7
Early (two weeks) post-operative appearance of a Syme's amputation stump.

A lateral radiograph of the heel, if not over-exposed, will often reveal this fundamental structure of the subcutaneous tissue. The vertical septa of elastic connective tissue are readily seen running from the skin below to the calcaneum above, or to the plantar aponeurosis more anteriorly (Fig. 14).

It is important to preserve this specialised subcutaneous tissue in the heel flap of a Syme's amputation. If the heel flap is dissected through the subcutaneous plane, the loculi are opened and their content of fat is forced out by pressure because they are no longer closed spaces.

FIG. 8

Vertical transverse section through foot, the plane of which corresponds approximately to the incision for Syme's amputation. Fat has been stained red to contrast with the white septa of fibro-elastic tissue which enclose the loculi of the heel pad.

In order to preserve intact this specialised weight-bearing subcutaneous tissue it is necessary that the plantar aponeurosis and the periosteum of the inferior surface of the calcaneum be removed intact with the heel flap.

THE IMPORTANCE OF SUBPERIOSTEAL DISSECTION OF THE HEEL FLAP IN SYME'S AMPUTATION

Syme elaborated the technique of his amputation with great care. Two incisions, the dorsal one to open into the ankle joint and the plantar incision carried directly through to the bones of the tarsus, outlined the extent of the heel flap. The talus was dislocated downwards and forwards from the mortise of the ankle joint and the calcaneum was freed from the tendo calcaneus and the heel pad by dissection with a sharp, stout knife in a plane which hugged the bone (*i.e.*, in the subperiosteal plane). In advocating this plane Syme was attempting to avoid injury to the calcaneal branches of the posterior tibial artery. That is important,

but a greater accomplishment is the assurance that the subcutaneous tissue with its special weight-bearing qualities will be included intact in the heel flap (Figs. 3 to 7).

Such a flap contains the stumps of origin of the short plantar muscles. It is a clumsy, untidy flap and every meticulous surgeon instinctively desires to tidy it up by removing the bulky muscle stumps (Alldredge and Thompson 1946). But to do so is likely to result also in the removal of the plantar aponeurosis. If this is done the subcutaneous loculi are opened and the fat is squeezed out. They can no longer function as hydraulic buffers.

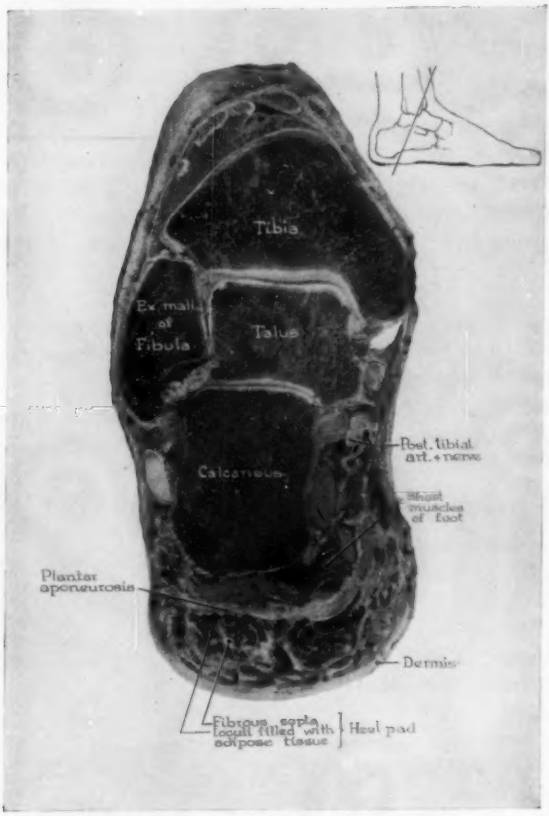

FIG. 9

The anatomy of the field of Syme's amputation to show especially the position and structure of the heel pad. This figure is based on Figure 8. Insert shows the plane of section.

Subperiosteal dissection of the calcaneum from the heel flap has another advantage. It leaves the heel flap lined with periosteum which more readily adheres to the cut surface of the tibia, and more firmly. Sometimes, indeed, new bone forms from this periosteal lining of the heel flap and this ensures very firm fixation of the heel flap to the tibia and an intact weight-bearing mechanism (Figs. 15 and 16).

It is of interest to quote an observation of Jacobson's (1889) which confirms this point. He described the technique of removal of the calcaneum from the heel flap by an approach from above. " The foot being still more pressed " (*i.e.*, downwards to dislocate the talus from the ankle joint), " the upper nonarticular surface of the os calcis comes into view and then the tendo achillis. This is severed and the heel flap next dissected off the os calcis from

above downwards, special care being taken to cut this flap as thick as possible; not to score or puncture it, but rather to peel it off the bone with the left thumbnail kept in front of the knife aided by touches of this." To this is appended a footnote: " If, in a young subject, the epiphysis comes away in the heel flap, it may remain there if the parts are healthy. The same

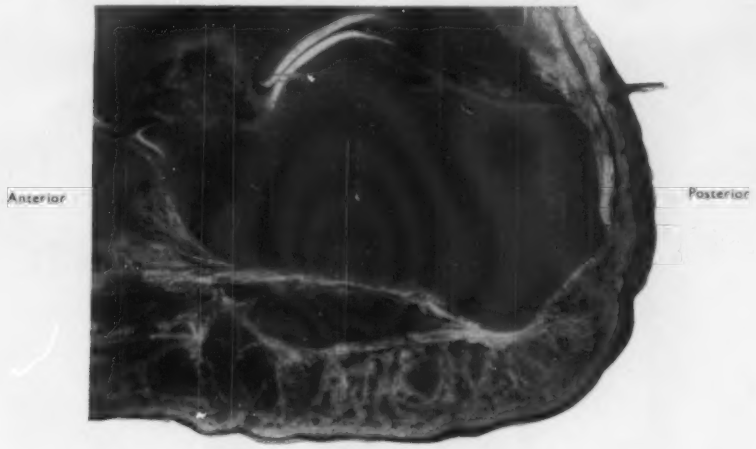

FIG. 10

Sagittal section through the heel of a seventeen years old girl to show the structure of the heel pad. Fat has been stained red in contrast to the white septa of dense elastic connective tissue which run from the dermis below to the plantar aponeurosis and the tuberosity of the calcaneum above.

FIG. 11

Vertical transverse section through the heel of a young patient to show the structure of the heel pad.

course may be followed with the periosteum, if it is found loose and peels easily away. Mr Johnston Smith, when amputating both feet for frost bite, left the periosteum on one side, on the other no attempt was made to save it. The first stump was much larger than the other, harder and more rounded; more like that of a Pirogoff's amputation."

This quotation is from the 1889 American edition of Jacobson's *The Operations of Surgery* and hence antedates the introduction of the Roentgen ray. In all respects, save the radiographic picture, his description indicates clearly the advantages of subperiosteal separation of the heel flap from the calcaneum.

We may conclude, then, that subperiosteal separation of the calcaneum from the heel flap is important for the maintenance of an intact weight-bearing covering of the stump and for the firm anchorage of the flap to the lower end of the tibia. It is unwise to tidy up the stump. This may damage the weight-bearing mechanism of the subcutaneous tissue and certainly will remove the periosteal lining of the heel flap and invite an unstable end to the stump.

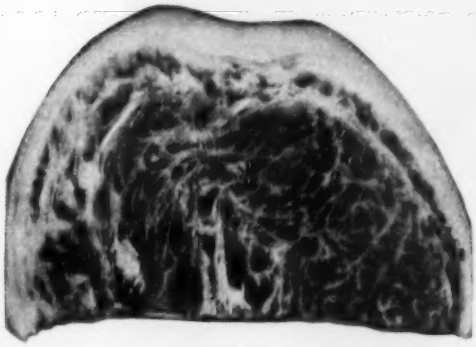

FIG. 12

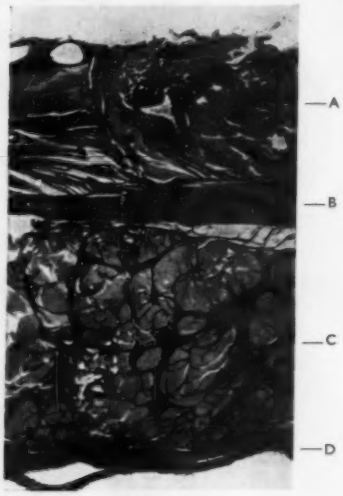

FIG. 13

Figure 12—Horizontal section through heel (parallel to the sole of the foot and below the level of the calcaneum) to show the structure of the heel pad. The septa and the loculi they enclose have been cut across at right angles to their vertical axis. The skin of the heel bounds this section medially, posteriorly and laterally. Figure 13—Thick frozen transverse vertical section of heel pad. (×2.) Stained with Oil Red 0 for fat and counterstained with light green. *From above downwards*—A—bellies of short muscles of foot; B—plantar aponeurosis; C—specialised elastic adipose tissue; D—skin.

IMPERFECTIONS WHICH MAR THE FUNCTION OF A SYME'S AMPUTATION STUMP AND HOW TO AVOID THEM

Not all Syme's stumps are perfect. Nevertheless most of the imperfections can be eliminated by meticulous attention to the technique of the operation and some can be compensated by the fitting and use of the prosthesis. In addition to damage to the heel flap the following are the most important faults which may prevent the achievement of a perfect result.

Misplaced heel flap—Care must be taken to place the heel flap beneath the tibia in such a manner that the plantar surface of the flap is exactly beneath the centre of the cut lower end of the tibia, and it must be maintained there until sound healing has occurred. This requires painstaking care because the heel flap is a large deep cup which fits loosely over the lower end of the tibia. At the end of the operation the correct position can best be secured by adhesive strips (Fig. 6). These should be inspected frequently during the first two weeks after operation to ensure that the correct position is maintained. If necessary they should be adjusted to keep the heel flap in its proper position. If the heel flap is improperly placed on the end of the tibia the margin of the cut surface of the lower end of the tibia will press through the skin against the inside of the prosthesis and cause discomfort (Fig. 17).

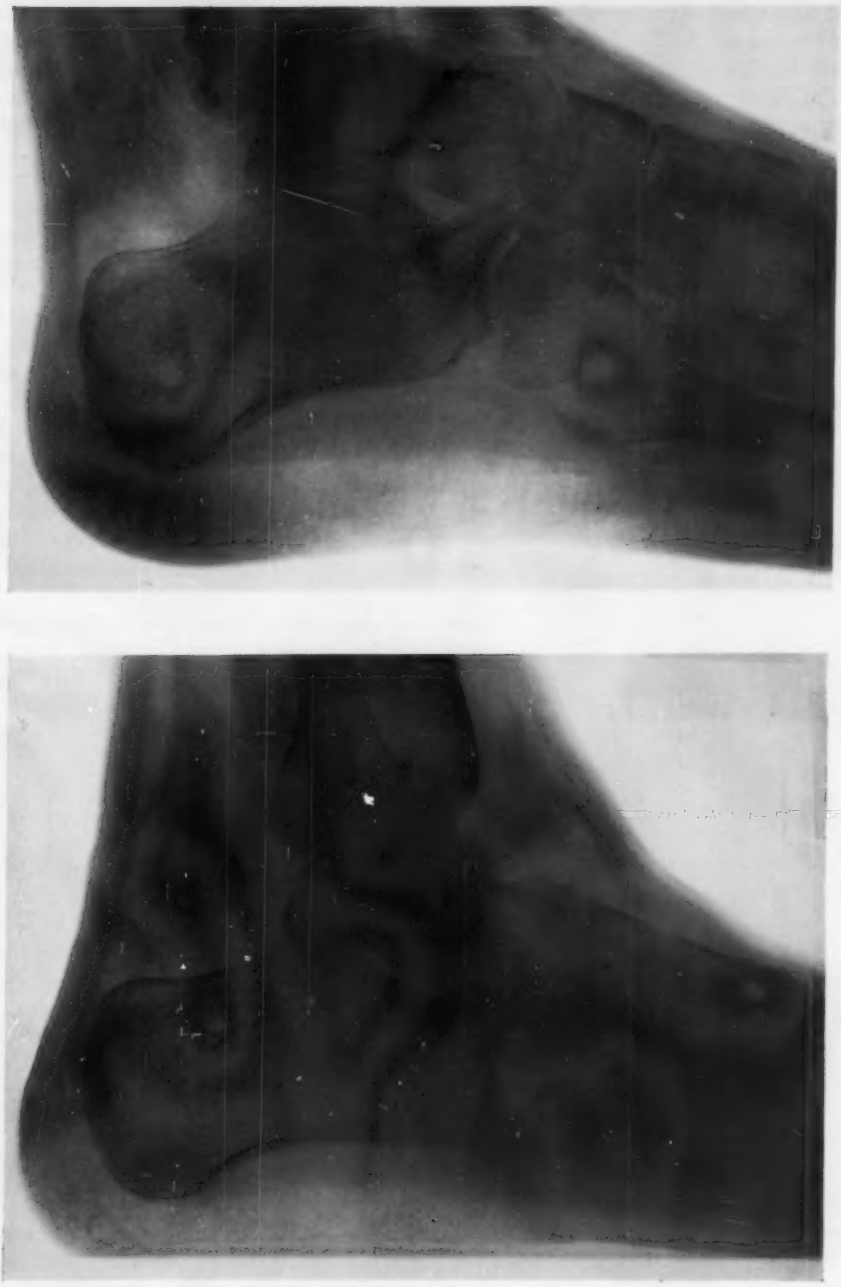

FIG. 14

Lateral radiographs of the foot to show the fibro-elastic septa of the heel pad when no weight is borne on the heel (*top*), and when the patient's weight is transmitted through the heel.

Sloping cut surface of the lower end of the tibia—When weight is borne upon the cut surface of the tibia which is not parallel to the ground the heel flap tends to be pushed to the high side of the slope. To avoid this the lower end of the tibia must be transected parallel to the ground, both in the transverse and in the antero-posterior plane. This does not necessarily mean at right angles to the long axis of the shaft of the tibia. For example, when the leg is bowed, the plane of section of the lower end of the tibia must be parallel to the ground, not at right angles to the long axis of the lower half of the tibial shaft.

Too small cross-section to cut end of tibia—The largest possible cross-section area of the cut end of the tibia and fibula is desirable. Smaller areas may result in localised pressure from weight bearing and consequent callus formation. Therefore the plane of transection should be just above the articular cartilage of the lower end of the tibia, to ensure the greatest possible area for the support of the heel flap.

" Wobbly " or unstable heel flap—If the heel flap is loose and easily displaced the pressure from use of the prosthesis may wipe it to one or other side or backwards. The bone edge then presses upon the scar. A flaccid, loose heel flap can be prevented by subperiosteal dissection of the heel flap. The deep surface of the heel flap then attaches itself firmly to the cut surface of the bone and the firm intact pad of adipose subcutaneous tissue resists changes in its shape. An unstable heel flap can be avoided only by proper technique. Once it has occurred it cannot be corrected by further operation, though its shortcomings can be minimised by modifying the lacing of the prosthesis (Fig. 17).

FIG. 15

Small mass of bone laid down in the heel flap of a Syme's amputation, the result of subperiosteal removal of the calcaneum. Useful in ensuring firm fixation of the heel flap to the tibia.

Tender heel flap with calluses—This is almost always due to failure to preserve the specialised elastic adipose tissue. It is accentuated if the cross-section of the cut end of the tibia is small or has projecting spurs. It can be prevented by proper fashioning of the heel flap during the operation. If the weight-bearing structures of the heel flap have been damaged by the operation their function cannot be restored by any subsequent operation.

Neuroma of the posterior tibial nerve—Careful preservation of the full thickness of the heel flap leaves the posterior tibial nerve in the flap. At the primary operation no attempt should be made to dissect this out and divide it at a higher level lest this cause damage to the weight-bearing qualities of the heel pad. If the neuroma which inevitably develops on its end becomes painful, late transection of the nerve at a level above the ankle joint without removal of the distal segment cures the condition.

Marginal gangrene of the heel flap—Except in cases of peripheral vascular disease this is nearly always due to faulty operative technique. Either the blood supply is impaired in the preparation of the flap by injury to the posterior tibial artery or the dressings are put on too tightly, or swelling occurs beneath the adhesive strips and they are not removed soon enough. With care in operating there is little danger of necrosis of the flap. Should necrosis occur, the stump is not necessarily ruined unless the loss of tissue is very great.

Vascular insufficiency in the heel flap—It has been said that the great length of a Syme's stump results in vascular insufficiency which is manifested by a cold, blue, painful end to the stump,

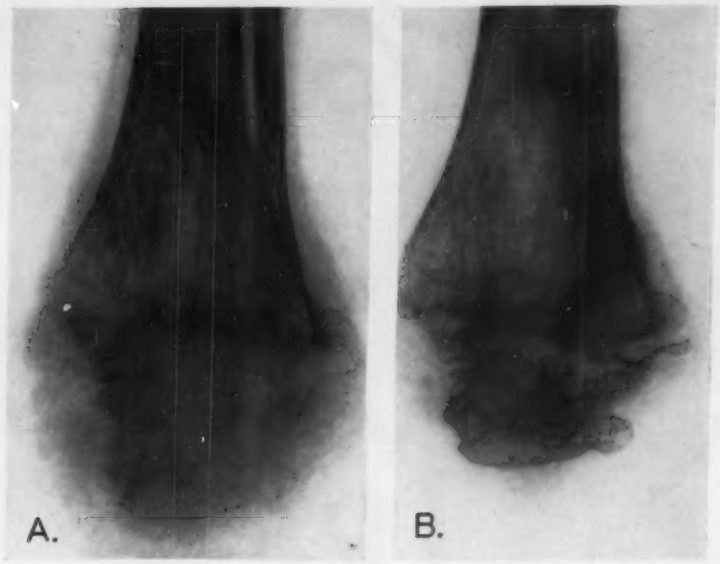

FIG. 16

Unusually large mass of bone laid down in the heel flap of a Syme's amputation stump. A) Four months after operation. B) One year after operation. This cloud of new bone is unusually large and heavy because osteogenesis was stimulated by the inflammatory reaction to tuberculosis of the tarsus for which the amputation was undertaken.

FIG. 17

FIG. 18

Figure 17—Misplaced and unstable heel pad on Syme's stump of left leg, the result of tidying up the heel flap by removing the stumps of the short plantar muscles, and with them the plantar aponeurosis and the periosteum of the calcaneum. The result is a heel pad imperfectly fused to the end of the tibia and in bad position. *Left*—With the muscles at rest and the heel pad held in place under the tibia by elastic traction. *Right*—When the peroneal muscles contract they drag the heel flap to the outer side of the stump.
Figure 18—Radiograph of the poor Syme's stump illustrated in Figure 17. In addition to the unstable heel pad, the high level of transection of the tibia provides a small area of support. In spite of these defects the stump has functioned reasonably well for nine years.

FIG. 19

FIG. 20

Figure 19—A functionally good Syme's stump. The heel pad is firmly fixed to the lower end of the tibia in good position. The area of support is broad. This amputation was undertaken to remove a foot much distorted after many attempts to correct recurrent club foot. Figure 20—Radiograph of the satisfactory Syme's stump shown in Figure 19.

FIG. 21

Standard Syme's prosthesis. A front lacing leather bucket moulded to the stump is fastened to a strong steel frame with an ankle block at the lower end; anterior and posterior views.

much accentuated in cold weather. This has not been the experience in Canada where many of our patients, in winter, are subjected to very low temperatures. Our experience would lead us to feel that vascular stasis from exposure to cold is not a problem of any importance.

It may be, however, that long-continued exposure to damp cold can produce vascular problems with which we are not familiar. Certainly chilblains are almost unknown to us while they are common in Great Britain. If this is the explanation of any vascular problem which may arise in Syme's amputation the cure may well be in sympathectomy rather than re-amputation at a higher level. A paralytic lower extremity after poliomyelitis in Canada

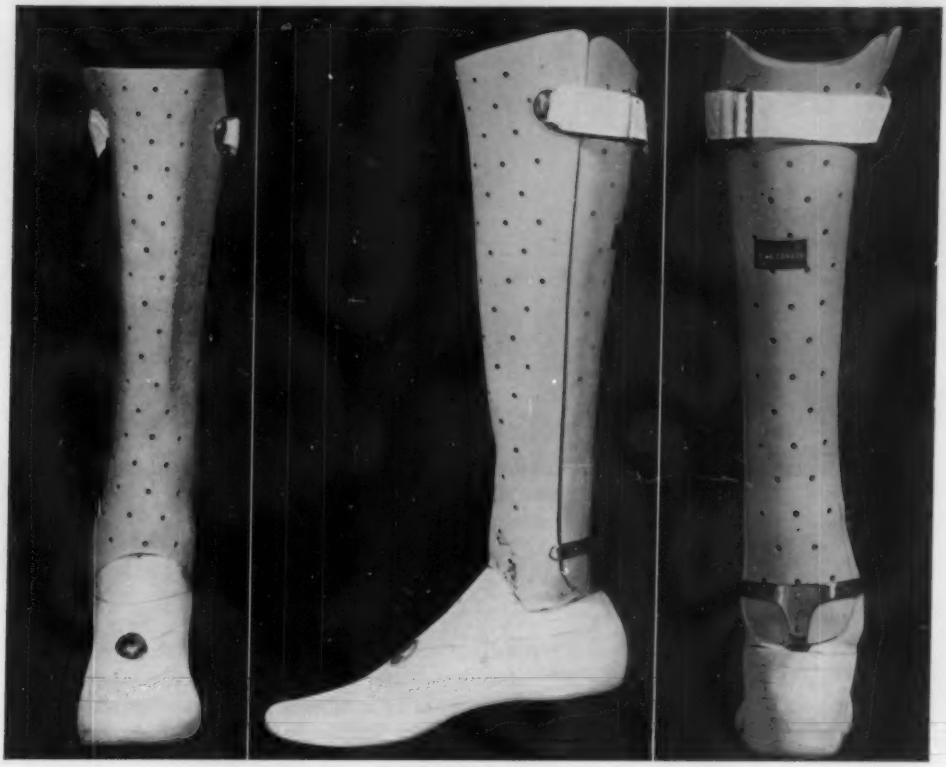

FIG. 22

Laminated synthetic resin bonded prosthesis for Syme's amputation made of layers of fibreglass cloth moulded over a plaster model of the stump and bonded with a rigid epoxy resin. The opening for the insertion of the stump is posterior and is closed by a plastic door buckled into place.

is often the source of much discomfort in cold weather. It becomes blue, cold, and painful and sometimes develops trophic skin lesions. This problem is completely relieved by sympathectomy.

SUMMARY

The details in technique which are most essential to ensure a perfect Syme's stump are the provision of a broad area of support for the heel flap by transecting the tibia and fibula as low as possible; the maintaining intact of the specialised weight-bearing qualities of the heel flap; and the proper placement of the heel flap under the cut ends of the tibia and fibula. If these aims are achieved a good and useful stump is assured; if they are neglected the stump

will be imperfect and may be unsatisfactory and no further operation can restore the qualities of the heel flap which are lacking.

It must be recorded, however, that Syme's stumps which are not technically perfect often function so well that there has been no need to consider re-amputation. A loose heel pad can be held beneath the end of the bone by firm lacing of the corset of the prosthesis. If its area of bony support is reasonably large it may serve well, though not perfectly, as an end-bearing stump. Syme's stumps so completely unsatisfactory as to necessitate re-amputation

FIG. 23

Figure 23—Double Syme's amputation. This man has worked for thirty-five years without interruption as an artificial limb maker. Figure 24—Double Syme's amputation shown in Figure 23 with prosthesis applied.

FIG. 24

have been those in which the plane of transection of the tibia is so high that the area supporting the heel flap is too small; or the weight-bearing qualities of the heel flap have been damaged; or there is instability of the heel flap which cannot be controlled; or there is impairment of nutrition of the heel flap.

INDICATIONS FOR SYME'S AMPUTATION

With a technique which ensures a satisfactory end-bearing stump, Syme's amputation is indicated in all destructive lesions of the foot provided that the skin of the heel is in good condition. The conditions for which it is most frequently performed are the following.

Severe injuries of the foot such as compound and comminuted fractures of the tarsus and metatarsus or crushing injuries of the foot. If it is evident that the injury to the foot is so severe that much of the foot will be lost or that the foot will become grossly deformed and rigid, a Syme's amputation should be performed as soon as the risk of infection is eliminated. With antibiotics available the amputation can sometimes be performed as a primary measure. More frequently severe contusion of the soft tissues plus actual or potential infection in the open wound will necessitate performance of the amputation as a secondary procedure after the wound has healed, or is reduced to a small size. In dealing with injuries of the foot, especially war injuries, the advantages of Syme's amputation should be borne in mind so that a two-stage operation can be performed rather than immediate resort to a mid-tibial

amputation. The primary stage will remove the shattered and infected foot, preserving the heel flap. The second stage, performed after infection is controlled or even after the wound of the foot has healed, will be a formal Syme's amputation.

Intractable infections of the bones and joints of the foot—At the present day this is less often an indication for Syme's amputation than formerly. Antibiotics enable us to master most pyogenic infections and even tuberculous infections respond well to streptomycin and iso-nicotinic acid hydrazide. A few unusual infections of the tarsus such as blastomycosis and Madura foot, which do not respond well to conservative treatment, can still be effectively treated by resection and the development of a Syme's stump. Syme's original operation was for tuberculous infection of the talus and calcaneum.

Deformities of the foot which cause serious disablement from rigidity and localised pressure. The chief cause of such deformities is previous trauma or infection, but such conditions as old club foot with intractable deformity can also be well treated by Syme's amputation.

Selected cases of obliterative vascular disease—Contrary to expectation it has proved feasible to deal with some cases of Buerger's disease and the occasional case of arteriosclerotic vascular disease by Syme's amputation. The most suitable case is in the young or middle aged man who has obliterative vascular disease with gangrene of the forefoot and a favourable response to lumbar sympathetic block. In such cases a lumbar sympathectomy followed by Syme's amputation will often give a good and useful stump which will last for years. Fifty per cent of our Syme's amputations for obliterative vascular disease have been successful. This is an important group in which function is much greater than would have been the case had their amputation been at a higher level, especially with the possibility that the other leg may need to be amputated later.

Frost bite and immersion foot—The problem here is similar to that in obliterative vascular disease, but less difficult. The vascular changes in the foot induced by cold cause thrombosis in the vessels of the involved area which can result in gangrene of the forefoot. Such cases are well treated by Syme's amputation.

Certain neurological problems such as neuropathic joints from tabes and syringomelia; intractable ulceration of the forefoot from irreparable sciatic nerve injury; spina bifida.

Malignant disease of the forefoot is an occasional indication for Syme's amputation.

SHORTCOMINGS OF SYME'S AMPUTATION

Syme's amputation has one shortcoming. It gives a bulbous-ended stump and is unsightly. The prosthesis must be bulky to accommodate the stump and it must be strong to resist the great stresses that are transmitted through it. Hence the patient is condemned to a large and heavy prosthesis, unattractive in appearance (Fig. 21). Some improvement has been accomplished by making the prosthesis of resin-bonded laminated fabric (Fig. 22). Even so the bulky ankle area is still obvious. It is not likely ever to be popular with women because of its appearance, but for men it is an excellent amputation.

SUMMARY AND CONCLUSIONS

1. Syme's amputation is a valuable amputation because it provides an end-bearing stump capable of taking full body weight and of transmitting well the stresses of locomotion.

2. The best end-bearing in a Syme's stump will be obtained when the area of bone supporting the heel flap is as broad as possible and when the specialised weight-bearing structure of the heel flap is maintained undamaged.

3. Technical steps in the operation which are essential for success are: *a*) Transect the tibia and fibula just above the articular cartilage at the lower end of the tibia. This gives the largest possible area of support. *b*) The line of transection must be parallel to the ground when the patient stands, not necessarily at right angles to the long axis of the tibia. *c*) The heel flap

must be separated from the calcaneum by subperiosteal dissection. This ensures the preservation of the weight-bearing elastic adipose tissue in the heel and firm attachment of the heel flap to the lower end of the tibia. *d*) The heel flap must be precisely placed beneath the tibia and held there until it is soundly healed.

My thanks are due to my friend and colleague, Gordon MacIntyre Dale, who for thirty-five years has been an ardent exponent of the merits of Syme's amputation. He has freely shared his experience with me. I am indebted to the Royal College of Surgeons of England for the photographs of Syme's knife and of the cast of his hand holding the knife. These are in the Hunterian Museum. My appreciation is due also to the Department of Prosthetic Services of the Canadian Department of Veterans' Affairs for their interest and skill in developing the specialised prosthesis for Syme's amputation and for their permission to include illustrations of the prosthesis in this paper. Professor J. C. Boileau Grant of the Department of Anatomy, University of Toronto, prepared in his Department the splendid specimens which illustrate the anatomy of Syme's amputation and the structure of the heel pad. Dr W. B. Anderson of the Department of Surgical Pathology, Toronto General Hospital perfected the technique which demonstrates clearly the histology of the heel pad (Fig. 13). Their assistance has ensured adequate illustration of this article. For this I tender them my grateful thanks.

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TENDON TRANSPLANTATION IN THE PREVENTION OF FOOT DEFORMITIES AFTER POLIOMYELITIS IN CHILDREN

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Since Nicoladoni's original description (1881) tendon transplantation has been used extensively in the treatment of paralytic foot deformities, but many of the results have been disappointing.

Whitman (1903) urged that tendon transplantation should be combined with stabilisation, and this seems to have been the usual practice in America. Two recently published series (Kuhlmann and Bell 1952, Reidy, Broderick and Barr 1952) recorded a high proportion of combined procedures.

By his work on physiological tendon transplantation, Mayer (1916) did much to establish the principles of tendon surgery. He advised, in the case of the foot, that if more than three muscles were paralysed, tendon transplantation was unlikely to be effective. Lange (1930) on the other hand, claimed to be able to produce a stable foot if only three muscles remained.

The electrobasographic studies of Schwartz, Trautmann and Heath (1936) have demonstrated how the various leg muscles contract automatically in different phases of the normal gait. Scherb (1952) has similarly investigated the action of transplanted muscles and has shown why many transplants fail to function. Generally a transplant will function automatically only if its normal phase of contraction corresponds with that of the muscle it is replacing.

These authors were concerned more with the active or automatic function of the transplanted muscle than with deformity of the foot, though all are agreed on the necessity of correcting fixed deformity before proceeding to transplantation. Goldthwait (1896), however, pointed out that the first result of a tendon transplantation was the removal of the deforming factor.

Tendon transplantation without tarsal arthrodesis has been performed sporadically at the Royal National Orthopaedic Hospital or its predecessors for many years: the earliest operations followed up in this review were performed in 1899. Since 1946 the idea that progressive deformity might be prevented by tendon transplantation has taken a firm hold. The authors were invited to undertake this independent assessment of the results as a guide to future policy.

CLINICAL MATERIAL

One hundred and forty-seven cases of tendon transplantation without bony stabilisation, 132 from the Royal National Orthopaedic Hospital, and fifteen from the Hospital for Sick Children, have been re-examined. In all cases the onset of poliomyelitis was in childhood and operation was undertaken before the age of fifteen years. The period of follow-up varied from six months to fifty-four years, with an average of three years.

TYPES OF DEFORMITY AND PATTERN OF PARALYSIS

Serious deformity seldom develops in a flail foot. Deformity is usually the result of muscle imbalance. At first strong muscles overcome their weak antagonists and produce postural deformity. Later, soft-tissue contracture and remodelling of bone lead to structural deformity. This is likely to be more severe in the growing foot.

The type of deformity is closely related to the pattern of paralysis. Table I shows the correlation between deformity and the important features of the paralysis pattern. The term " medial cavus " is used to designate those feet with invertor weakness which do not develop

a plano-valgus deformity. Depression of the first metatarsal, from weakness of the tibialis anterior, leads to an increase in the medial longitudinal arch of the foot. On active dorsiflexion the deformity is increased, with pronation of the forefoot and dorsiflexion or clawing of the big toe.

SURGICAL TECHNIQUE

Although a large number of different procedures were used, the technique generally followed the principles laid down by Robert Jones (1911): 1) Operation should seldom be undertaken before the age of five years and only after at least a year's conservative treatment. 2) Structural deformity should be corrected before transplantation is undertaken. 3) Tendons should be rerouted subcutaneously in as straight a line as possible. 4) The transplanted tendon should be reattached to bone. In reinforcing the calf the transplanted tendons have usually been reinserted into the tendo calcaneus.

In operations to restore muscle balance two main techniques may be used: 1) reinforcement of weak antagonist muscles; and 2) transplantation of the main deforming muscle so that it may serve a useful function. It is often possible to combine both these techniques.

TABLE I

RELATIONSHIP OF PARALYSIS TO DEFORMITY

Deformity	Main muscle weakness
Calcaneo-cavus .	Calf alone
Calcaneo-cavo-valgus	Calf and invertors
Calcaneo-cavo-varus	Calf and evertors
Medial cavus . .	1. Tibialis anterior alone 2. Tibialis anterior and posterior
Equino-plano-valgus	1. Tibialis anterior and posterior 2. Tibialis anterior alone
Equino-varus . .	1. Peronei alone 2. Peronei and dorsiflexors

RESULTS

The results are presented in Table II. In assessing them the main criterion has been the presence or absence of deformity, rather than the voluntary or automatic function of transplanted muscles. In many of the cases classified as unaltered it is possible that operation has prevented a further progression of the deformity; and included among those classified as worse are a number of calcaneus feet in which the deformity was overcorrected. This is often an acceptable end-result.

To be classed as improved by the transplant the foot should show less deformity. Correction of fixed deformity is an essential preliminary to transplantation, and most of this correction should be maintained.

FAILURES

The cases rated as worse are analysed in Table III. This shows that failure may be due to increased deformity, overcorrection, instability, or a secondary deformity attributable to the transplant.

Increased deformity may be due to failure to correct fixed deformity before transplantation: this applies particularly to the tight tendo calcaneus. Technical faults such as insecure fixation of the transplanted tendon, and badly planned operations, explain some of the failures.

Overcorrection, instability and secondary deformity are best considered in relation to the three systems of balance in the foot.

First system: dorsiflexion-plantarflexion—The dorsiflexors are the tibialis anterior and the long extensors; the plantarflexors are the calf muscles and peroneus longus and the long flexors of the toes.

TABLE II

RESULTS OF TENDON TRANSPLANTATION (147 operations)

Deformity	Operative procedure	Improved	Unaltered	Worse	Total
Calcaneo-cavus	Peronei to calf	2	3	2	7
	Peronei and other posterior muscles to calf	5	3	2	10
	Other transplants	—	1	1	2
Calcaneo-cavo-valgus	Peronei to calf	11	3	6	20
	Peronei and other posterior muscles to calf	--	2	2	4
	Peronei and anterior muscles to calf	1	1	1	3
	Peronei to dorsum	1	—	2	3
Calcaneo-cavo-varus	Tibialis anterior to calf	2	—	—	2
	Posterior muscles to calf	1	1	—	2
	Tibialis anterior reinserted on dorsum	2	2	—	4
Medial cavus 1*	Extensor hallucis longus to first metatarsal or cuneiform	6	5	—	11
	The same combined with other transplants	1	1	4	6
	Peronei to dorsum	4	1	4	9
Medial cavus 2*	Extensor hallucis longus to first metatarsal	1	2	1	4
	Peronei to dorsum	2	1	1	4
	Multiple transplants	1	2	2	5
Equino-plano-valgus 1*	Peronei to dorsum	7	7	2	16
	Peroneus longus to dorsum	1	4	—	5
	Other transplants	—	—	4	4
Equino-plano-valgus 2*	Peronei to dorsum	2	2	—	4
Equino-varus 1*	Tibialis anterior to dorsum	10	4	2	16
	Other transplants	2	1	—	3
Equino-varus 2*	Tibialis posterior to dorsum	1	1	—	2
	Tibialis posterior and toe flexors to calf	—	1	—	1
Totals		63	48	36	147

* See Table I

In cases of calcaneus deformity an attempt has usually been made to reinforce the calf. Reinforcement of a totally paralysed calf can seldom, if ever, balance a strong tibialis anterior. In such cases tibialis anterior, as a deforming factor, should be transferred to the calf or to the mid-dorsum of the foot. On the other hand, reinforcement of a paretic calf, when tibialis

TABLE III

ANALYSIS OF FAILURES

Case number	Age at onset	Years between onset and operation	Years between onset and follow-up	Operative procedure	Deformity increased	Over-correction	Instability	Secondary deformity	Comment on result
				Deformity: Calcaneo-cavus					
6	2	1½	2	Peronei to calf	—	—	—	Varus	Invertors strong
7	1	3¾	1	Peronei to calf	—	—	—	Varus	Invertors strong
16	3	2¼	1	Peronei, long flexors and tibialis posterior to calf	—	Equinus	—	Varus	Transplants acting as tenodesis
17	2	4½	1	Peronei and long flexors to calf	—	—	+	Varus	Invertors strong
19	6	1¾	2	Peronei to calf. Two and a half years later extensor hallucis longus and peroneus tertius to calf	—	—	+	—	—
				Deformity: Calcaneo-cavo-valgus					
34	10	½	5½	Peronei to calf	—	Equinus	—	Plano-valgus	Calf underestimated
35	3	1	3	Peronei to calf	—	Equinus	+	—	Calf underestimated
36	1	1¾	2	Peronei to calf	—	—	+	—	All muscles weak
37	1	2	2	Peronei to calf	—	Equinus	—	Plano-valgus	Tibialis anterior: 0
38	3	3	3	Peroneus brevis to calf	+	—	+	—	Tibialis anterior: 4
39	3	3	3	Peronei to calf	—	Equinus	+	—	—
42	¾	3	1½	Peronei, tibialis posterior and flexor digitorum longus to calf; extensor hallucis longus moved	—	—	+	—	—
43	4	3	1¾	Peronei, tibialis posterior and flexor digitorum longus to calf	—	Equinus	—	Plano-valgus	Calf quite strong before transplant
46	2	2¾	2¾	Peronei to calf. Nine months later, extensor hallucis longus and tibialis anterior to calf	—	Equinus	+	—	—
48	4	2½	2	Peronei to dorsum (navicular)	+	—	—	Varus	Calf overestimated
49	3	3¼	1	Peroneus longus to dorsum	+	—	+	—	—
				Deformity: Medial cavus 1*					
71	1	2	2¾	Extensor hallucis longus to tibialis anterior. Nine months later: peroneus longus to calf. One year later: extensor digitorum longus to tibialis anterior	—	—	—	Varus	Excessive reinforcement of tibialis anterior, with a tight tendo calcaneus
72	1	3	1	Extensor hallucis longus to tibialis anterior. One and a half years later: extensor hallucis longus and extensor digitum longus to first metatarsal, peronei to calf	—	—	—	Plano-valgus	—
73	1	4½	3¼	Extensor hallucis longus to tibialis anterior, peronei to calf. Six months later: extensor digitum longus to tibialis anterior	—	—	—	Varus	Peronei to calf, but tibialis posterior = 4

* See Table I

TABLE III—(*contined*)

Case number	Age at onset	Years between onset and operation	Years between onset and follow-up	Operative procedure	Deformity increased	Over-correction	Instability	Secondary deformity	Comment on result
				Deformity: Medial cavus 1*—*continued*					
74	2	4½	2½	Extensor digitorum longus to tibialis anterior. One and a half years later: peroneus longus to calf	—	—	—	Plano-valgus	Tight tendo calcaneus
80	11	¾	2½	Peroneus longus to tibialis anterior	—	—	—	Varus	Peroneus longus reinserted too far medially
81	1	2	1¾	Peronei to second cuneiform	—	—	—	Varus	Tibialis posterior underestimated
82	1	2½	1½	Peronei to second cuneiform	—	—	—	Varus	Tibialis posterior underestimated
83	5	2¾	1½	Peroneus brevis to tibialis anterior	—	—	—	Plano-valgus	Tight tendo calcaneus
				Deformity: Medial cavus 2*					
91	2	3	1½	Extensor hallucis longus to first metatarsal	+	—	—	—	Tendo calcaneus tight and attachment of transplant slipped
95	2	3	1½	Peronei to second cuneiform	—	—	—	Plano-valgus	Tight tendo calcaneus
99	2	2	3	Extensor digitum longus and peroneus tertius to tibialis anterior. Peroneus longus to calf	—	—	—	Plano-valgus	Tendo calcaneus tight and attachment slipped
100	3	10	1¾	Extensor hallucis longus to tibialis anterior, extensor digitum longus 4 and 5 and peroneus tertius to mid-dorsum. Peronei to calf	—	—	—	Plano-valgus	Tight tendo calcaneus
				Deformity: Equino-plano-valgus 1*					
115	1	2½	1	Peronei to tibialis anterior	—	+	—	Varus	Transplant too far medial
116	1	5¾	4	Peronei to first cuneiform	—	+	—	Varus	Transplant too far medial
122	7	1¼	2¼	Peroneus brevis to tibialis anterior	+	—	—	—	Ineffective transplant when tendo calcaneus tight
123	¾	1½	1½	Peroneus brevis to tibialis anterior	+	—	—	—	
124	3	3	4¾	Extensor hallucis longus and peroneus tertius to tibialis anterior	+	—	—	—	—
125	1	4	1½	Extensor hallucis longus to tibialis anterior and peronei to calf. Six months later: extensor digitorum longus 4 and 5 and peroneus tertius to extensor digitorum longus 2	+	—	—	—	—
				Deformity: Equino-varus 1*					
140	¾	2½	2½	Tibialis anterior to peroneus tertius	—	+	—	Valgus	Transplant too far lateral
141	1	2¾	4	Tibialis anterior to fifth metatarsal, extensor hallucis longus to first metatarsal	—	+	—	Valgus	Transplant too far lateral

* See Table I

anterior is weak, can and frequently does lead to a secondary equinus, with severe plano-valgus in some cases.

When the calf is strong there is usually some fixed equinus associated with a varus or valgus deformity. Failure to correct this contracture will cause persistence of the varus or valgus.

Second system: inversion-eversion—The main muscles concerned are the tibialis posterior and the peroneus brevis. Tibialis anterior and peroneus longus are of secondary importance. The stability of the foot depends largely on this system.

When the calf is weak and the tibialis posterior and peroneus brevis are well balanced, as in a calcaneo-cavus deformity, it is unwise to transplant one of these muscles and leave the other, because secondary varus or valgus may follow. Transplantation of both may lead to instability.

With a strong calf and a varus deformity it is possible to balance the overacting tibialis posterior by transplanting the tibialis anterior towards the outer side of the dorsum of the foot. When the heel is in valgus it is impossible surgically to counteract a strong peroneus brevis, if tibialis posterior is paralysed. In such cases it is usual to transplant both peronei to the dorsum of the foot, but it is important to be sure that the tibialis posterior is really weak before doing this, otherwise a secondary varus may follow.

Overcorrection of a valgus heel can occur even when the tibialis posterior is paralysed, if the peronei are transplanted to the inner border of the dorsum. It is safer to reattach them in line with the second metatarsal. Similarly overcorrection of a varus heel will usually follow transplantation of tibialis anterior to the cuboid or fifth metatarsal; reinsertion in line with the fourth metatarsal is preferable.

Third system: pronation-supination (or depression and elevation of the first metatarsal)—The main muscles are the peroneus longus and the tibialis anterior, but the flexors and extensors of the big toe are also important. If tibialis anterior is weak, extensor hallucis longus can depress the first metatarsal by dorsiflexing the big toe. With weakness of peroneus longus the long and short flexors of the hallux may contribute to elevation of the first metatarsal.

Depression of the first metatarsal is likely to follow transplantation of tibialis anterior if extensor hallucis longus is strong. If the tendo calcaneus is tight this deformity may become troublesome. Conversely, a strong tibialis anterior will elevate the metatarsal if peroneus longus is transferred to the calf; in such cases tibialis anterior should also be moved.

DISCUSSION AND CONCLUSIONS

The principles of tendon transplantation have been laid down by many authorities; this study has served to underline the importance of some of them.

1. Operation should not be undertaken until at least a year after the onset of the disease. This rule has been broken on three occasions in this series; in two cases the result was spoilt by further recovery of muscle power.

2. It is preferable to delay operation until the age of five years: firstly, because accurate muscle charting may be difficult in a young child; and secondly, because overcorrection and secondary deformities occur more readily. On the other hand the primary deformity may progress more rapidly in the young child, making earlier operation sometimes justifiable.

3. Accurate muscle charting is essential. An apparently paralysed muscle may recover considerable power after correction of fixed deformity; similarly a transplantation to assist the weak muscle may result in its gaining power. For this reason a period of physiotherapy should follow correction of a fixed deformity before transplantation is planned.

4. Structural deformity must be corrected so far as possible before transplantation is carried out. This applies particularly to a tight tendo calcaneus. When the limb is short or the quadriceps weak, some degree of fixed equinus may be desirable; but if it is decided not to correct the equinus, too much must not be expected of a transplant to restore side-to-side balance.

5. The hazards of operation are overcorrection, instability, and secondary deformity. In designing an operation the foot as a whole should be considered in relation to the three systems of balance, lest in restoring balance in one system one destroys it in another. In cases of extensive paralysis it is impossible to achieve both balance and stability by tendon transplantation alone.

When weakness is not severe the following procedures are fairly well established. 1) Transplantation of tibialis anterior towards the outer border of the dorsum of the foot for equino-varus deformity from weakness of the peronei. 2) Transplantation of extensor hallucis longus to the first metatarsal, preferably with interphalangeal arthrodesis, for " medial cavus " from isolated weakness of tibialis anterior. 3) Plano-valgus deformities may be treated by transplantation of the peronei to the dorsum of the foot, but the results are less reliable. It is possible that in some cases further progression of the deformity is prevented, but in the more severe deformities tendon transplantation alone is inadequate. 4) The results of calf reinforcement are even less predictable; they range from complete failure, through satisfactory results, to equino-valgus from overcorrection, or gross instability. If there is a calcaneo-cavus deformity with side-to-side balance, tendon transplantation is of doubtful value. On the other hand, in some cases of calcaneo-cavo-varus or valgus the muscles causing the varus or valgus may profitably be transplanted to the tendo calcaneus even at the risk of producing an equinus deformity.

The results presented here are mostly early. In analysing them we have considered mainly the deformity of the foot; strict standards of assessment have been adopted, and the pitfalls of tendon transplantation have been emphasised.

Some of the operations giving poor results as regards deformity have nevertheless improved function. The method as a whole has a place in the treatment of the partly paralysed foot in the child.

Conclusion—A carefully planned operation may be expected to check increasing deformity without doing harm, and to make subsequent bony stabilisation easier. In favourable cases it may be possible to restore muscle balance and stability, making further surgery unnecessary. A longer follow-up is necessary to determine to what extent this ideal can be achieved.

We wish to thank all those surgeons who have allowed us to examine their cases. We thank particularly Mr H. J. Seddon and Mr K. I. Nissen for their help and encouragement.

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PARALYTIC SCOLIOSIS *

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Every human endeavour should have, for convenience, both a definite beginning and clear cut limits. This essay is no exception. It starts from the classical paper of Colonna and Vom Saal in 1941. It is not proposed to review the literature before this date; this paper is concerned only with consideration of advances in knowledge that have been made since then—indeed the only justification for writing it is the belief that advances in knowledge have been made.

By paralytic scoliosis I mean scoliosis occurring in patients who have had a definite attack of poliomyelitis. This paper refers only incidentally to scoliosis due either to other disorders of the nervous system or to myopathy; and it does not consider cases of so-called idiopathic scoliosis that might be due to poliomyelitis but in which there is no definite evidence of the patient's having had the disease. It is not intended to discuss, except incidentally, the vexed question of the etiology of idiopathic scoliosis, even though electromyography suggests that some cases are really due to localised muscle paralysis.

Definitions. *Trunk muscles*—These include all muscles acting on the trunk which may be concerned with posture. It is customary to differentiate spinal and extra-spinal muscles. The former can be separated into the superficial longitudinal group whose main function is extension and, to a lesser extent, lateral flexion; and a deep oblique and transverse group whose main function is rotation and, secondarily, lateral flexion. The extra-spinal group includes the abdominal, quadratus lumborum, psoas, intercostal, rhomboid, trapezius and latissimus dorsi muscles. Electromyography shows that all these muscles participate in the maintenance of posture and that the postural function of the extra-spinal muscles becomes important if the spinal muscles are weak or paralysed.

By *convex* to the right I mean that the vertebrae at the apex of the major curve are shifted and their bodies rotated to the right. The *major curve* is usually the primary or determining one, other curves being secondary or compensatory. It is usually the greatest in both lateral angulation and rotation. It is usually the most fixed and arises first in time; occasionally it is hard to say which curve is primary and two equal curves may arise simultaneously. In paralytic cases this usually occurs only in Type II (general collapse).

INCIDENCE

Colonna and Vom Saal (1941) found that out of 600 patients with poliomyelitis 150 developed scoliosis. They did not state how many of these were mild cases in which the spinal deformity was of little or no clinical significance. In most clinics the incidence of severe scoliosis after poliomyelitis is much less than 25 per cent. Indeed, we now recognise that infection with a neurotropic virus usually manifests itself in the form of an abortive or non-paralytic attack; most authorities consider that nearly all adults have had at least a mild infection with one of the poliomyelitis viruses. In recent epidemics it has been recognised that non-paralytic and abortive cases far outnumber classical paralytic cases; therefore if, at the time of an epidemic, 600 cases of frank infantile paralysis are recorded, there must almost certainly have been many hundred times as many cases of undetected neurotropic virus infection. Therefore, the statement that 25 per cent of patients with poliomyelitis developed scoliosis cannot be either accurate or significant as it entirely depends on a somewhat arbitrary definition of both poliomyelitis and scoliosis. The exact figure is not of any great practical

* Robert Jones Prize Essay 1954.

importance except in so far as there is always the chance of a child who has had poliomyelitis developing a scoliosis—even years afterwards. Therefore, a careful watch must be kept on the back of every poliomyelitis patient.

Steindler (1929) found that out of a total of 700 cases of scoliosis 31 per cent were due to poliomyelitis. The exact criterion of scoliosis was not mentioned. If all mild postural curves were included I think the incidence of paralytic cases would be much lower.

Scott (1950) found that in a series of 321 cases of scoliosis at Oxford only nineteen were due to poliomyelitis.

In a small personal series twenty-three out of forty-eight cases of severe scoliosis were definitely due to poliomyelitis, but it is only fair to state that this was a selected series, composed mainly of patients with severe curves who were referred for further investigation.

On the other hand, many cases labelled " idiopathic scoliosis " may really be paralytic; so the true incidence of paralytic cases might be much higher. The exact figure is not very important; what does matter is that in every case of scoliosis a careful search should be made for evidence of a previous attack of poliomyelitis.

ANATOMY

It is not proposed to give a detailed review of the anatomy and physiology of the spine as there are already several excellent accounts (Lovett 1931, Kleinberg 1951). The factors which preserve the normal alignment of the spine are complex. The action of the various trunk muscles, the shape of the bones, the attachments, size and direction of ligaments, and the inherent rigidity of such structures as the intervertebral discs all participate. In general, the spine with its supporting trunk muscles may be compared with a jointed flagpole held upright by multiple elastic and contractile guy-ropes that radiate in all directions. As elsewhere in the body there is normally considerable physiological reserve, and a number of the " guy-ropes " could be cut without the flagpole's tilting over; but if more than a certain number are cut, and especially if the cut ones are adjacent, the pole collapses. As elsewhere, muscle imbalance is more likely to produce deformity than would the same degree of paralysis if distributed evenly. Of course, the analogy is not exact and there is some intrinsic stability in the normal adult spine from the shape of the bones, the tension of the ligaments and the turgor of the intervertebral discs. In the adult spine fairly extensive paralysis can occur with relatively little deformity; but in the child the bones are soft and plastic, ligaments are elastic and the intervertebral discs are less turgid. Thus the younger the patient the greater the deformity that a given degree of muscle paralysis is likely to produce. Moreover, once deformity is established in a child, secondary disturbance of growth may aggravate the deformity still more.

It is not proposed to give a detailed account of the clinical manifestations of scoliosis, the general features of which are well known; clinical descriptions will be given only where it is thought that they would help to emphasise some special point.

The curves of paralytic scoliosis are usually said to be longer, more mobile, less acute, less severe and associated with less vertebral rotation than idiopathic or congenital curves. This is often so but it is not necessarily always the case. Many cases of paralytic scoliosis have been seen in which the curve is severe, fixed, short and sharp, and in which rotation is marked. In general, paralytic curves conform to the above classical description in their early stages; but if progressive deterioration occurs they become radiologically indistinguishable from idiopathic curves (Fig. 1).

Paralytic scoliosis is associated with two main disabilities. Firstly, there is the spinal deformity and associated secondary deformities of thoracic and abdominal parietes and contents. Secondly, there is the spinal instability. The two disabilities do not necessarily run parallel. Some patients have severe deformity but relatively little weakness, and the problem of treatment is essentially the same as for idiopathic scoliosis—namely to prevent

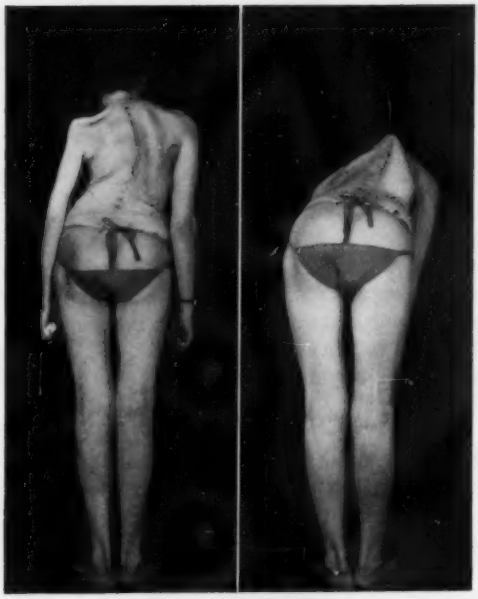

FIG. 1
Case 1—Primary thoracic (Type 4) curve. Clinical photographs before treatment.

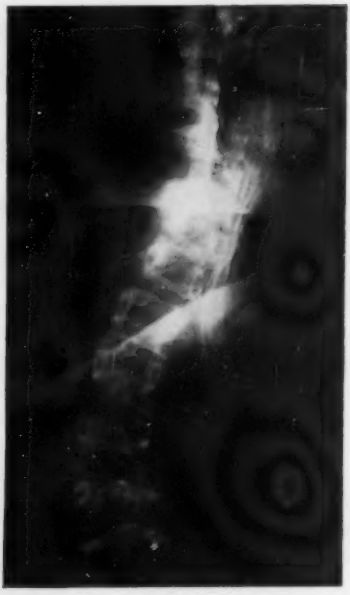

FIG. 2 FIG. 3
Case 1—Primary thoracic curve. Figure 2—Radiograph before operative correction. Figure 3—After operative correction.

and correct deformity. In these, as in idiopathic scoliosis, the first step is to try to discover whether the deformity is progressive or not.

In other cases there is extensive weakness but relatively little fixed deformity, and the problem is how the spine can best be strengthened by such measures as external supports, bone grafting or muscle development. There is an infinite variety of combinations but in every case the two aspects—deformity and instability—must be considered. This great variety in the clinical picture makes classification difficult.

CLASSIFICATION

It has been found convenient and helpful to recognise four main types of deformity.

1. General thoraco-lumbar C-curve. This usually occurs in patients with relatively slight paralysis who have been allowed up too early. Rotation deformity is slight. In recumbency the deformity is usually slight but it becomes more marked on standing (Figs. 8 and 9).

FIG. 4

FIG. 5

Case 2—Primary thoracic (Type 4) curve. Figure 4—Radiograph before operative correction. Figure 5—After operative correction.

2. General " collapse " type or combined thoracic and lumbar curve. This is usually due to extensive spinal weakness. The curves are mobile, at any rate for a considerable period, and head suspension usually produces marked correction. Rotation deformity is moderate (Figs. 10 to 17 and 20 and 21).

3. The primary lumbar curve due to a combination of pelvic obliquity and imbalance of the trunk muscles. There is usually a compensatory thoracic curve of less degree which is secondary in character (Figs. 18 and 19).

4. Primary thoracic curve. This is often associated with weakness of the scapular muscles and there are secondary compensatory curves above and below (Figs. 1 to 7).

Primary cervical curves are not considered here because no definite paralytic cases with fixed deformity have been seen. It is, at any rate, a theoretical possibility that primary cervical scoliosis from unilateral paralysis of the neck muscles in children might occur. I have only seen such cases in adults and in them fixed deformity does not occur. Presumably the cases

in which the cervical muscles are widely affected are usually of the " brain-stem " type which has carried a high mortality in the past. It will be interesting to see whether, with improved methods of treatment of this type of case and a higher survival rate, more cases of primary cervical scoliosis will be seen.

ETIOLOGY

At first sight the mechanism of deformity in paralytic scoliosis should be obvious. One would expect that the muscles on the stronger side would pull the spine over to that side—that is, the spine should be convex to the weaker side, much as in the foot an inversion deformity tends to develop if the evertors are weak. However, the opposite is usually the case. Colonna and Vom Saal (1941) found that in 70 per cent of cases the curve was convex to the stronger side and in only 20 per cent was the curve concave to the stronger side. These latter were all cases with marked imbalance of the abdominal muscles. Unfortunately, they did not

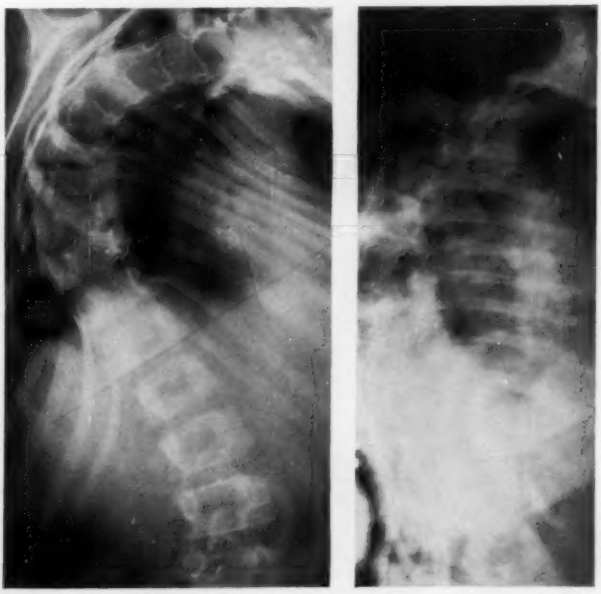

FIG. 6 FIG. 7

Case 3—Primary thoracic (Type 4) curve. Figure 6—Radiograph before operative correction. Figure 7—After operative correction.

differentiate between the different types of curve and did not give details; so it is not possible to deduce the exact mechanism of deformity from their paper.

The following factors have been suggested as being responsible for deformity in paralytic scoliosis: pelvic obliquity, imbalance of abdominal, psoas, quadratus lumborum, diaphragm, intercostal, rhomboid, trapezius, latissimus dorsi and deep and superficial spinal muscles. In trying to decide the relative importance of these various factors certain observations have been made which are set out below, and the possible significance of each one of the above factors will be considered in greater detail.

Pelvic obliquity—This factor has been much stressed by Lowman (1932), Mayer (1936) and others. It can undoubtedly be an important contributory factor in producing lumbar scoliosis. At the same time, one sees large numbers of cases of pelvic obliquity due either to disease of the hip joint such as tuberculosis, congenital shortening of one leg or poliomyelitis, in which there is only slight curvature of the spine and the spine is fully mobile and well compensated.

In other words, if the trunk muscles are normal fixed severe scoliosis does not occur; so I think it is clear that pelvic obliquity is not the primary cause of scoliosis, although it may be an important contributory cause.*

Abdominal asymmetry—It has been suggested that weakness of, for example, the right abdominal muscles will inevitably produce a scoliosis with convexity to the right, just as weakness of the rectus abdominis muscles usually produces a lordosis. Here again the facts do not entirely fit this theory. One sees quite a large number of patients with gross unilateral weakness of abdominal muscles without any scoliosis and in some cases of lumbar scoliosis electromyographic investigation shows increased tone and activity of the lateral abdominal muscles on the convex side. Nevertheless, asymmetry of the abdominal muscles can be an important contributory factor as shown by three cases of injuries to the spinal cord occurring in boys in their early 'teens. In each case, after a period of spinal shock, tone, reflex activity

FIG. 8

FIG. 9

Case 4—Thoraco-lumbar general C-curve.

and spasm returned sooner on one side of the body than on the other and in each case there was a marked lumbar scoliosis with the concavity towards the stronger and more spastic muscles; the scoliosis was much improved after subarachnoid injections of alcohol had been given to paralyse the spastic muscles. In these cases it is probable that other muscles besides the abdominal muscles were also in spasm. Nevertheless it can be concluded that gross imbalance of the abdominal muscles can be an important contributory factor in lumbar scoliosis but it cannot produce scoliosis if the other trunk muscles are normal. Sometimes the scoliosis is convex to the stronger side; in such cases imbalance of the spinal muscles is the dominant factor.

Psoas muscle—The psoas muscle has also been suggested as an important factor in scoliosis. It is, however, not uncommon to see patients in whom one psoas muscle is acting and the

* There is a special type of thoraco-lumbar scoliosis associated with lumbo-sacral obliquity but this is usually congenital not paralytic and is not considered here.

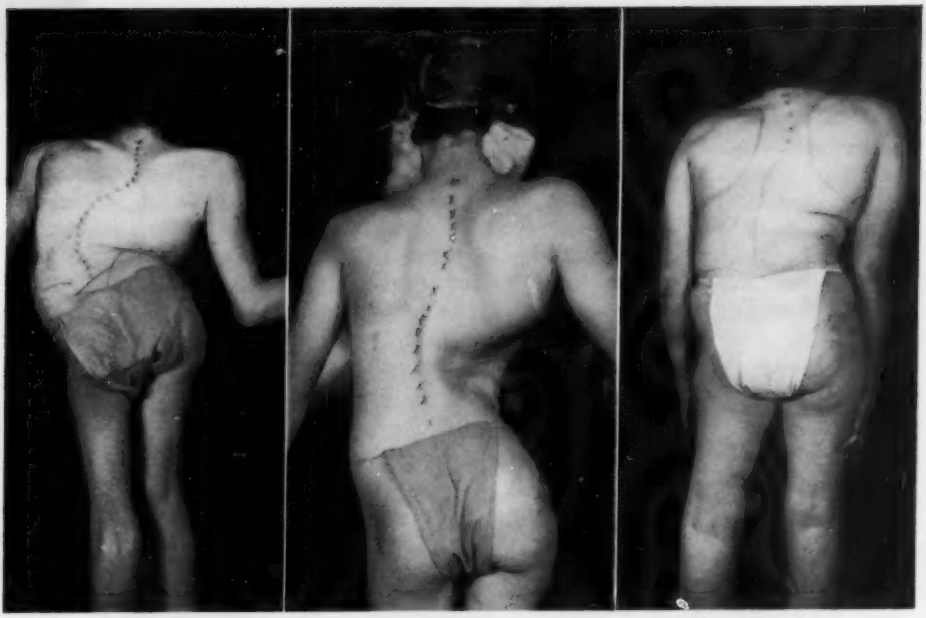

FIG. 10 FIG. 11 FIG. 12

Case 5—" General collapse " combined thoracic and lumbar (Type 2) curve. Figure 10—Standing before treatment. Figure 11—With head suspension before treatment. Figure 12—After treatment.

FIG. 13 FIG. 14

Case 5—" General collapse " combined thoracic and lumbar curve. Figure 13—Radiograph before treatment. Figure 14—After spinal fusion.

other is paralysed but in whom there is no scoliosis. Thus it would seem that by itself the psoas muscle is not a very important factor, although it is true that if a marked flexion deformity of the hip occurs from the unbalanced action of the psoas muscle this may be associated with some scoliosis, and that attempts to correct the flexion deformity of the hip may aggravate the scoliosis unless the psoas tendon is lengthened. However, in general, my experience is that the psoas muscle is not a very important factor.

Colonna and Vom Saal (1941) found that in 103 cases the scoliosis was convex to the stronger side. In lumbar curves they attributed this to over-activity of the psoas muscle on that side but they had no means of estimating the activity of the deep spinal muscles and it seems probable that these are a more important factor. Even if the deformity is associated with imbalance of the psoas muscles this may be a coincidental rather than a causal factor. The psoas muscle and the spinal muscles often have the same segmental innervation and it must commonly be the case that the degree of paralysis in the psoas and spinal muscles runs parallel.

Quadratus lumborum—This cannot be easily tested clinically, but with electromyography it is possible to obtain a fair idea of its activity. In some patients increased tone of the quadratus lumborum appears to be a factor in producing a lumbar scoliosis convex to the opposite (weaker) side but these are not always patients who have had anterior poliomyelitis. However, in the true paralytic cases obvious imbalance of the quadratus lumborum usually appears to be associated with convexity towards the affected (weaker) side. Quadratus imbalance is a contributory rather than a primary factor.

Diaphragm—It has been suggested that unequal diaphragmatic activity is an important factor both in paralytic and in idiopathic scoliosis (Jansen 1913). For some time past all patients with paralytic scoliosis have been radiographed to estimate the range of diaphragmatic movement, and in no case has there been any suggestion of unbalanced diaphragmatic activity. Similarly, from a large thoracic unit only two cases of scoliosis could be found associated with unequal diaphragmatic activity. These were in children with diaphragmatic herniae and other congenital defects; so it is probable that the scoliosis was due to inherent growth disturbance and not to the unequal diaphragmatic activity. There is no real evidence that unequal diaphragmatic action is an important factor in producing scoliosis.

Intercostal muscles—It is difficult to test for activity of the intercostal muscles clinically but this can be done fairly easily by means of electromyography. In general, intercostal muscles have a slight inspiratory activity in quiet breathing. As the breathing becomes deeper the inspiratory activity increases; with forced respiration there is activity in expiration also. The main function of the intercostal muscles is to fix the ribs. In addition, the intercostal muscles play a part in maintaining normal posture, and such activities as lateral flexion and head lifting are accompanied by increased intercostal activity. It is not uncommon to find that the intercostal muscles on one side show greater activity at rest, on forced respiration and on movement. The muscles on the convex side nearly always show the greater activity and as this finding occurred in cases of scoliosis from a large number of different causes—congenital hemivertebrae, idiopathic, neurofibromatosis—there were grounds for believing that the increased intercostal activity is a secondary compensatory phenomenon and not a primary cause.

Scapular muscles—Colonna and Vom Saal (1941) stressed the role of the scapular muscles in producing thoracic paralytic scoliosis. It will be recalled that they found that 81 per cent of patients with upper limb paralysis developed scoliosis and that 7 per cent of their patients with scoliosis had no demonstrable involvement of the trunk muscles. They also found that twenty-one patients had demonstrable involvement of the trunk muscles but no scoliosis. They therefore tended to stress the importance of the extra-spinal muscles. It is hard to believe that the scapular muscles play an important role in producing gross scoliosis. It is a common experience that patients often have appreciable paralysis of the arm or shoulder girdle

without more than slight scoliosis. Similarly, in less than half the cases of thoracic paralytic scoliosis could gross scapular weakness be detected. My opinion is that the upper limb paralysis is coincidental rather than causal and is merely an expression of the fact that muscles with a common segmental nerve supply tend to be simultaneously affected in poliomyelitis. If Colonna and Vom Saal (1941) had had the facilities for performing electromyographic investigations they would probably have found involvement of the deep spinal muscle in all cases of thoracic paralytic scoliosis and I think they would have attached less importance to the extra-spinal muscles.

Longitudinal superficial erector spinae muscles—In the overwhelming majority of cases these showed increased tone and activity on the convex side both on clinical testing and on electromyographic examination. In a few cases the longitudinal superficial spinal muscles were weaker on the convex side (at the apex of the curve) but the deep oblique spinal muscles

FIG. 15

FIG. 16

FIG. 17

Case 6—" General collapse " combined thoracic and lumbar (Type 2) curve. Figure 15—Radiograph with patient standing before treatment. Figure 16—Standing with head suspension. Figure 17—Standing after spinal fusion.

were stronger. The deep oblique spinal muscles can only be tested with needle electrodes; unfortunately it is not really practicable to use needle electrodes in small children and we could not investigate fully some of the cases where the distribution of the paralysis was anomalous. The longitudinal superficial erector spinae muscles are usually considered both to extend the spine and to assist in lateral flexion. The muscles on both sides act synergistically—for example, on right lateral flexion the right muscles contract first but the total activity of the left muscles is nearly as great—in the normal subject. In cases of scoliosis from a number of different causes besides poliomyelitis the muscles of the convex side show increased activity both at rest and on extension and on lateral flexion. It is considered that this shows that in these cases they are not the primary etiological factor but that their increased activity is probably a compensatory phenomenon. Thus if a hemivertebra produces a scoliosis the muscles on the convex side will show greater activity in their efforts to hold the trunk upright.

Deep oblique spinal muscles—Whenever possible these were investigated with needle electrodes and tested electromyographically, but needle electrodes cannot easily be used as a routine

in small children. In general it was striking that the muscles on the convex side showed greater activity at all phases than those on the concave side. This suggested, therefore, that an important factor in the etiology of paralytic scoliosis is imbalance between the deep oblique spinal muscles, whose main action is to produce rotation of the spine. If muscle imbalance causes rotation of the spine towards the stronger side then the various clinical phenomena of a typical case of scoliosis will develop.

In order to explain the fact that scoliosis is usually convex to the stronger side five main theories have been put forward. The first, associated with the name of Steindler (1929), suggests that the curve is a compensatory phenomenon. The argument is as follows. If the centre of gravity were in the centre or shifted to the strong side the weak muscles on the

FIG. 18

FIG. 19

Case 7—Primary lumbar (Type 3) curve. Figure 18—Before operative correction. Figure 19—After operative correction.

opposite side would be unable to prevent the body from falling over to the stronger side. In other words, lacking the normal balancing action of the muscles on one side the body maintains its balance by shifting the centre of gravity. The same mechanism is seen in patients with a weak gluteus medius muscle—they lean over to the weak side. Just as in paralysis of the gluteus medius the centre of gravity is shifted towards the weaker side (otherwise the body would tend to fall over to the normal or stronger side) so, in paralytic scoliosis, the centre of gravity is shifted to the weaker side.

The argument is ingenious; it is certainly true that the stronger muscles are nearly always on the convex side. It is, however, hard to see how this mechanism could give rise to severe fixed deformity. (One does not usually see a fixed abduction contracture of the hip in cases of gluteus medius paralysis.) Also in paralytic scoliosis head-and-shoulder raising in the

prone position usually produces or aggravates the deformity, and the compensation mechanism is unlikely to be operating in recumbency.

In relatively mobile scoliosis in the thoraco-lumbar region with considerable instability compensation appears to be a factor, especially when the element of spinal weakness has not been recognised in the acute phase and too early ambulation has been allowed. In such cases a period of muscle development in recumbency usually produces a striking improvement. It seems, however, that, while compensation may be a contributing factor, it is unlikely to be the main factor in producing severe fixed scoliosis. The term compensation can also be applied in a different sense. In cases of lateral curvature of the spine from unequal vertebral growth (congenital or acquired) the spinal muscles on the convex side often show greater activity than those on the concave side. It is probable that this is a compensatory phenomenon, namely an unconscious or reflex effort to straighten the spine.

The compensation theory is partly true but fails to explain all cases. It appears to be true in most general C-curves where the whole body inclines to the weaker side and the centre of gravity is shifted to this side (Fig. 8). In my opinion it fails to explain the more localised progressive curves with compensatory curves above or below, and in which the centre of gravity is either not appreciably displaced or is displaced to the convex side.

Colonna and Vom Saal (1941) stressed the importance of the extra-spinal trunk muscles. Lateral abdominals, quadratus lumborum and psoas in the lumbar region, and the trapezius, rhomboid and latissimus dorsi in the thoracic region. Undoubtedly, these can play an important part and we have seen a number of cases in which imbalance of these muscles was associated with scoliosis convex to the weaker side, but we have seen a much larger number of cases in which the convexity was to the stronger side and also a large number of cases in which there was imbalance of these muscles without scoliosis, so this theory also is inadequate to explain all the phenomena. It should be pointed out that the mere fact that scoliosis may be associated with paralysis of certain extra-spinal muscles does not prove that these are the causal agents. Their paralysis may be merely an incidental expression of the fact that deep spinal muscles with the same segmental innervation are affected.

The third theory is that there is a primary disturbance of vertebral growth; at the present time there is no way of proving or disproving this theory. In severe scoliosis from any cause secondary growth disturbance with vertebral wedging frequently occurs but the question here is whether the growth disturbance in paralytic scoliosis is usually or ever the prime cause. It is, however, reasonable to point out that shortening in a "poliomyelitis leg" is not necessarily proportional to the extent of paralysis and it is possible that the involvement of the spinal cord includes nerve cells which have a direct or indirect effect on bone growth. These cases in which the scoliosis only manifests itself after a considerable latent period are most easily ascribed to the illness's causing a primary disturbance of vertebral growth. In view of the paucity of evidence it is hardly profitable to discuss this theory further at present except to point out that severe deformity may occur with only slight paralysis and that degree of deformity and paralysis do not run *pari passu*. Its applications for treatment and prevention are discussed later.

The fourth theory is that the main developing factor is imbalance of the deep oblique and transverse rotator muscles of the spine. Electromyography shows that these are nearly always stronger on the convex side and that there is often no sign of activity on the concave side. This theory explains the progressive and intractable nature of many cases of paralytic scoliosis. It also makes it possible to include many cases of idiopathic scoliosis in the paralytic category as many of these show similar electromyographic tracings. If this theory is true it opens up new possibilities of prevention.

Lastly, it has been suggested that fascial contracture causes scoliosis (Bennett 1952, Clark 1954). We do know that in certain situations such as the fascia lata fascial contracture may occur after poliomyelitis and may cause severe deformity of the hip. Unfortunately we

FIG. 20

FIG. 21

Case 8—" General collapse " combined thoracic and lumbar curves (Type 2) treated by Allan's jack method. Figure 20—Before treatment. Figure 21—After treatment.

FIG. 22

FIG. 23

Case 8. Figure 22—Radiograph before treatment. Figure 23—After treatment.

know very little about the reasons why fascial contractures sometimes occur after an attack of poliomyelitis and for the present this theory must be considered possible but unproven.

In such a complex condition as paralytic scoliosis it is unlikely that there will be one single adequate cause; at least five factors may be involved: 1) compensation; 2) imbalance of the extra-spinal muscles; 3) primary disturbance of vertebral growth; 4) imbalance of the deep spinal rotator muscles; 5) fascial contracture.

These factors may interact and either reinforce or counteract one another. This explains some anomalous clinical findings such as that a lumbar scoliosis is sometimes convex and sometimes concave to the side of the abdominal weakness. This is not a mere academic problem: if we knew more accurately the exact factors involved then prevention and treatment of the early case would be more effective.

PROGNOSIS

So far no comprehensive survey of the natural history of untreated paralytic scoliosis has been published similar to the papers published on the natural history of idiopathic scoliosis (Ponseti and Friedman 1950, James 1951, 1954). We know that many curves remain mild and unprogressive and that even severe curves may remain mobile for long periods. We know that in general the younger the age of onset the worse the ultimate deformity is likely to be. We know that latent periods may occur followed by rapid deterioration during a burst of rapid growth. We know that paralytic lumbar curves are more likely to be severe and disabling than idiopathic lumbar curves. It is clear that a great deal more work requires to be done on this problem and that until more is known about the natural history of paralytic scoliosis claims to have prevented deformity or deterioration must not be accepted uncritically.

PREVENTION

In the early management of poliomyelitis certain procedures are traditionally employed. **Rest**—Chronic overstrain of a temporarily paralysed muscle inhibits or delays its recovery; overstrain in the early stages acts similarly. In the past patients with poliomyelitis have often been kept in bed for long periods, but recently there has been a trend towards earlier ambulation and patients are allowed up sooner. This is sometimes undesirable, especially in children. I have seen a number of cases of early scoliosis due to children being allowed up too soon. These cases usually respond well to a period of rest followed by gradually increasing back exercises in recumbency. Before a child is allowed up after an attack of poliomyelitis its back should be carefully examined, and if there is any tendency to scoliosis complete electromyographic investigation should be done. It is only right, however, to point out that prolonged rest in the early stages will not always prevent scoliosis. Some patients develop severe deformity even in recumbency; and unduly prolonged recumbency may have adverse physical and psychological effects. Recumbency would have to be ridiculously prolonged to have any preventive effect in those puzzling cases of " late onset " scoliosis which develop after a latent period. Perhaps routine electromyography in the convalescent phase would cast some light on the problem of which patients are likely to develop deformity and what preventive measures should be instituted.

Active, assisted and passive movements are rightly considered of great importance for the prevention of deformity in poliomyelitis. Unfortunately, they are less effective in the spine than elsewhere. There are so many joints and muscles in the spine that it is hard to focus treatment on the particular ones which are mainly involved—for example, the deep rotator muscles. Severe scoliosis may develop during and in spite of the best physiotherapy. Furthermore, the spinal muscles act bilaterally and simultaneously—thus on right lateral flexion in recumbency against resistance the left spinal muscles are usually as active as the right ones and vice versa. Therefore, spinal exercises nearly always strengthen the muscles

which are already stronger. It is almost impossible to give spinal exercises which strengthen the weaker muscles. This is not to decry physiotherapy, which is a necessary and valuable adjuvant in all cases; but it is important to realise its limitations and that often something more must be done if scoliosis is to be prevented.

Splintage—Splints have been used in poliomyelitis for three main purposes: 1) to limit or prevent overstretching of paralysed muscles; 2) to prevent deformity by offering resistance to the unopposed pull of the unparalysed muscles; and 3) to support flail joints. It is no longer generally believed that it is necessary for a paralysed muscle to be continually relaxed. To obviate secondary joint stiffness and to prevent atrophy of unparalysed muscles rigid and prolonged splinting has been largely abandoned; instead either intermittent or " lively " splinting is commonly used. This form of splinting is, however, usually inadequate to counteract gross muscle imbalance such as frequently occurs in the foot and spine. No form of temporary, removable or " lively " splint known at present will prevent scoliosis if the tendency to deformity is strong; but some such splint may be useful in the convalescent period to give temporarily paralysed muscles the best possible chance of recovering. In the case of postural muscles early ambulation is contra-indicated.

A large variety of supportive splints and jackets have been devised to prevent fixed deformity from developing in paralytic scoliosis. All are relatively ineffective; most have the disadvantage of restricting respiratory movement and most do more harm than good. This does not mean that they should never be used but it does mean that they should only be used if they are clearly doing good, otherwise they are definitely doing harm. In general, unless an appliance or jacket is so extensive as to be hopelessly cumbersome it will not prevent progressive deformity if the tendency to deformity is marked. Night splints are often used in poliomyelitis to prevent deformity. To be effective they must fit well and be applied regularly and efficiently. This is difficult in the case of a growing and unruly child but in selected cases lateral plaster beds are useful.

If the spine is of the collapsing type (Type 2) some splintage is certainly required. Appliances such as the Milwaukee brace which support the head and the pelvis are effective but cumbersome, heavy and uncomfortable and may cause atrophy of the mandible. The skin of the trunk is not very tolerant of pressure and no external splint will correct or prevent deformity of the spine if the deforming force is very strong. Other appliances rely on axillary crutches, fixed to a rigid body jacket; in the very flail spine these are essential, but they are uncomfortable and may cause pressure paralysis of the nerves in the axilla. The " Taylor " brace appears to be the most useful for this type of paralytic scoliosis. The same effect can usually be obtained by making the patient use crutches—indeed it is rare for a patient who regularly uses crutches to develop a severe scoliosis. This simple but effective procedure in the prevention of scoliosis is not used sufficiently often. In recent years it has usually been considered desirable to try to teach children to walk without crutches whenever this is possible. If the child has a weak back this is a questionable policy. A jacket without either head extensions or axillary crutches is quite ineffective in holding up a collapsing spine, and by inhibiting respiration usually does more harm than good; but by making him use crutches a constricting jacket can often be avoided and progressive scoliosis prevented.

Prophylactic operations—From the point of view of preventing deformity a completely flail foot presents a much easier problem than the partly paralysed one, and to a large extent this is true in the case of the spine. In the last few years we have learned the value of early tendon transplants in the foot to prevent progressive deformity, especially of inversion or eversion. Tendon transplants cannot prevent all deformities; they are effective only in carefully selected cases in which muscle strengths can be assessed accurately before operation, otherwise correction may be either excessive or inadequate. If the muscles can be correctly balanced there is little or no tendency to deformity and it is then possible to omit splinting except in so far as this is necessary to support flail, weak, weight-bearing joints.

Are there any comparable procedures in the case of the spine? In lumbar scoliosis with abdominal weakness various operations with fascial slings have been advocated (Lowman 1932, Mayer 1936), but no large series of late results have been reported and I have no personal experience of such procedures in the treatment of scoliosis. They are of course certainly valuable to improve the "tone" of the abdominal wall. They do not seem to be widely practised in England; possibly they should be more widely used but personally I do not believe that abdominal weakness is often the main cause of severe spinal deformity.

In addition, transplanted fascia usually does not withstand a strong continuous muscle pull.

On theoretical grounds stripping the lateral insertions of the deep oblique spinal muscles from the transverse processes on the convex side should be a valuable procedure. At the same time, the transverse processes at the apex of the curve can be wired together with the intention of inhibiting growth on the convex side (Figs. 24 and 25).

FIG. 24

FIG. 25

Figure 24. Case 9—Radiograph after wiring of transverse processes on convex side and "stripping" of deep rotator muscles. Figure 25. Case 10—Another patient radiographed after intertransverse wiring.

Stapling to produce epiphysial arrest is a well-tried and successful procedure at the knee to correct genu valgum. It is, therefore, natural that stapling of vertebral bodies should have been tried for scoliosis. Some papers on the subject have been published (Nachlas and Borden 1950, Rieth *et al.* 1950) but it cannot be said that the results have been very striking so far. There are three main reasons for this: 1) the maximal growth asymmetry is postero-lateral (neural arch) rather than antero-lateral. 2) It is hard to make the stapling effective over a sufficient number of vertebrae. The effectiveness of epiphysial stapling depends essentially on the staples spanning a wide enough area so that when growth occurs in the growing area it is compressed. Obviously in the spine where a little growth occurs at a large number of different places a considerable number of these growth areas must be included in one staple if the pressure is to rise sufficiently for growth to be inhibited. 3) The mechanism of vertebral growth is not known for certain and possibly merely spanning the intervertebral disc does not affect the main growth centre.

As an alternative to inserting staples into the vertebral bodies wiring together several transverse processes on the convex side has recently been tried as a means of delaying vertebral growth on this side. The early results are encouraging but it is too early to assess the permanent value of the procedure.

Prophylactic bone grafting to prevent further deformity has been used extensively. In general, it may be said that the nearer the child is to the end of its growth period the more effective the result seems to be, but that spinal fusion will not prevent further deformity occurring in a young child with gross muscle imbalance. Bone grafting has usually been advocated as a support on the concave side. Once the transplanted bone is incorporated it becomes as pliant as the child's other bones: it is well known that in childhood the bones are pliable and become bent if subjected to a strong deforming influence; therefore if the deforming influence is still active a bone graft on the concave side in childhood will not prevent recurrence of the deformity. Bone grafts have also been used on the convex side to " tether " this side and inhibit further growth. An adequate study of the late results of bone grafts on the convex side has not yet been published.

It seems that rational preventive measures are hardly possible until our ideas of the mechanism of production of paralytic scoliosis are clearer. There is need for further investigation.

TREATMENT

Many cases of paralytic scoliosis are mild and are non-progressive. By no means all require active treatment; in many the spinal disability is relatively slight and in relation to other disabilities may be insignificant, but all cases require observation and investigation and if deterioration occurs active measures to prevent further deterioration should be taken without delay.

A crooked spine with its serious secondary effects on the thoracic and abdominal viscera is obviously a very considerably added disability in a child whose muscles are already weak; so a marked scoliosis should always be corrected if this can be done without harm to the patient. In poliomyelitis it is a platitude that one treats the individual, not the disease—a platitude easily said and even more easily forgotten.

In a given case of paralytic scoliosis there are a large number of factors to be considered—age, sex, temperament, courage, general health, intellectual and scholastic abilities, manual skill, social background, parents' co-operation, condition of arms and legs. Before deciding that an attempt should be made to correct the deformity all these factors must be assessed. It can be said at the outset that correction is always difficult; it may involve the child's being away from his normal environment for a long time. After correction the child's muscles will be weak and a long period of muscle building and rehabilitation will be needed. Before embarking on treatment one should be reasonably certain that the improvement will be worth while. Nothing is worse than long periods of hospitalisation, immobilisation in plaster and multiple operations if there is no worthwhile improvement at the end. It is a mistake to try to treat the deformity unless the treatment is going to be effective.

Many patients with poliomyelitis have mild non-progressive deformities of the spine which do not require special treatment. In others, the general condition is too poor or the paralysis too extensive to allow of any very ambitious treatment. In another group correction of the deformity may improve the sitting posture but diminish the power of locomotion. Each case must be judged on its own merits and the decision to attempt correction should be taken only after assessing every factor.

Before starting to correct the deformity secondary factors such as pelvic obliquity, short leg, flexion-abduction of hip, should be corrected first. Here again there is a problem; after correction of the pelvic obliquity the general posture may appear worse.

If the curve is relatively slight and mobile, and the trunk muscle imbalance is not too

great, correction of the pelvic obliquity* will improve the scoliosis. On the other hand, if the scoliosis is severe and fixed and there is marked trunk muscle imbalance correction of the pelvic obliquity will make the posture seem worse—until or unless the scoliosis is corrected (Figs. 26 and 27). It is better not to start on the pelvic obliquity unless one is confident that the lumbar curvature can be corrected.

In order to correct severe pelvic obliquity—with, for example, abduction, flexion, lateral rotation contracture of the right hip and adduction-flexion contracture of the left hip with subluxation or even dislocation—it is best to correct the adducted left hip first. Divide the tight adductors and, if necessary, perform an extra-pelvic obturator neurectomy, and if the hip remains dislocated either perform an osteotomy or resect the head and neck of the femur. Then apply a plaster spica to the trunk and *right* leg: apply a plaster cylinder to the left leg and, with the pelvis fixed by the spica, gradually (over two or three weeks) swing the left leg into an overcorrected position. Then apply a new spica to the trunk and left leg which is now in neutral position. The next step is to lengthen the right iliotibial band by multiple fasciotomy, apply a plaster cylinder to the right leg and swing it slowly over towards the left leg until the deformity is overcorrected. If the right hip has become ankylosed in the abducted position it will be necessary to perform either a corrective osteotomy or a resection of the head and neck of the femur. The legs are now parallel and at right angles to the interspinous line; in other words if the real shortening is corrected the pelvis will be level.

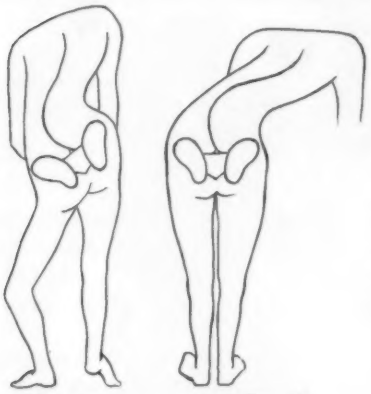

FIG. 26 FIG. 27

Case 11—Lumbar scoliosis and pelvic obliquity with abducted left hip and adducted right hip. Figure 26—Before correction of pelvic obliquity. Figure 27—After correction of pelvic obliquity but before correction of the lumbar scoliosis. The legs are parallel, the pelvis is level but the posture is worse.

Until recently correction of spinal deformities has usually been attempted by either applying a plaster jacket or using continuous traction and lateral slings. A plaster jacket may be applied in flexion or extension or on a special frame such as that devised by Abbott (1911). Windows may be cut or the plaster may be divided and hinges and turnbuckles applied. In all the variations of this method the principle is the same—continuous or intermittent external pressure on the thoracic cage to mould the spine into shape. Obviously the external force acts only indirectly on the spine through the medium of skin, ribs, etc. There is always a risk of pressure sores, or the thoracic cage may become even more deformed, so that a "therapeutic" deformity is added to the one already present.

Nowadays most surgeons who attempt correction by plaster jackets use some form of "Risser" jacket (1948). Alternatively the patient may be placed in bed with the head of the bed raised; "chin-occiput" and shoulder straps are applied and fastened to the head of the bed. The patient also wears a pelvic belt to which a 15–20 lb. weight is attached by cords running over pulleys at the foot of the bed. Broad webbing binders are now applied round the patient's body and attached to weights which hand over pulleys attached to the side of the bed.

The advantage of this arrangement is that the slings can be released for physiotherapy, back and breathing exercises. The disadvantage is that the patient must spend most of the day

* The term "pelvic obliquity" is used in two senses. Firstly, to describe a structural twist in the bony structure of the pelvic ring. This is of obstetrical rather than of orthopaedic significance, it is not amenable to any known form of treatment. Secondly, the term is used to denote tilting of the pelvis secondary to fixed deformity of one or both hips. This type is amenable to surgical treatment and is the type which is discussed here. Both types may exist together. Lumbo-sacral obliquity is a separate entity.

in recumbency. There is probably little to choose between the two methods in effectiveness*; whatever method is used the limiting factor is the skin's tolerance of pressure.

In poliomyelitis the curve consists essentially of a central relatively fixed portion with relatively mobile portions above and below. Although considerable apparent correction is often obtained, this is usually at the mobile transitional extremities of the curve, not at the central fixed position (Figs. 28 and 29). There is, therefore, a considerable tendency to recurrence after the jacket is removed, and unless an extensive spinal fusion is performed the deformity usually relapses to worse than it was previously. After a spinal fusion external support is still necessary until the fusion has consolidated; this may take nine to twelve months. Even after consolidation in young children the bone is plastic and recurrence of the deformity may occur. The greatest successes of " turnbuckle " correction and fusion are in the "general C " type of curve where the spine is relatively mobile and the chief need is for support rather than correction (Figs. 10 to 17). In the " general collapse " type, extensive spinal fusion is the best treatment. Fortunately, the introduction of bone banks has made the

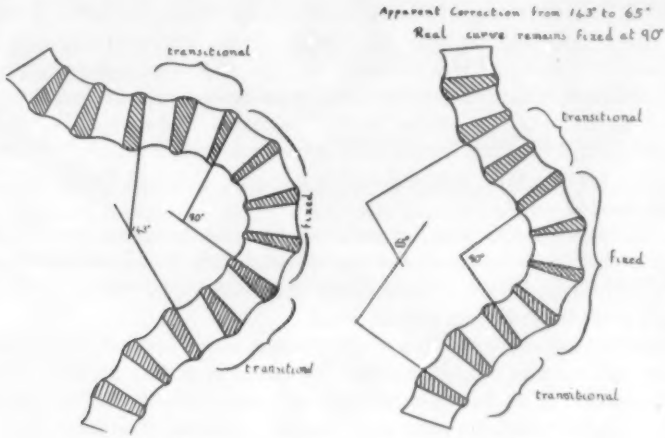

FIG. 28 FIG. 29

With correction by conservative measures the central fixed portion of the curve remains unchanged and there is a strong tendency to relapse unless extensive spinal fusion is performed. Figure 28—Before correction. Figure 29—After correction.

operation much less shocking and few patients need to be denied its benefits. Great care is needed in deciding how low the area of spinal fusion should occur. Many patients, especially those with weak hips or those who have to use calipers, depend on mobility of their lumbar spine. They " walk " by lifting first one and then the other side of their pelvis. Treatment which unduly stiffens the lumbar spine and especially the lumbo-sacral joint may decrease the power of walking. This may be justifiable if they sit with greater comfort but it should never be done lightly.

Recently two new methods of obtaining correction have been introduced. While they are still too recent for long-term assessment they show considerable promise in certain types of case. **Allan's method** (1954)—A metal " jack " is inserted between transverse processes at each end of the curve on the concave side; the " jack " is opened and as much correction as possible is then obtained. Chips of cancellous bone are packed round it to hold the spine in its corrected position. The method is most successful with fairly mobile lumbar curves. With fixed rigid curves in the thoracic or thoraco-lumbar region the transverse processes may break. In the

* In the combined thoracic and lumbar curve (Type 2) the second method is the more effective.

lumbar region all motion is abolished—this may make walking harder but will improve the sitting posture (Figs. 20 and 21).

Roaf's method (1954)—A wedge of bone including laminae, pedicles, transverse processes and adjacent portions of two bodies and intervening disc are resected; the gap is then closed, thus correcting the deformity. After correction the spine may or may not be stable; if it is not, spinal fusion will be required at a later date. The exact indications for this procedure have not yet been defined (Figs. 1 to 7 and 18 and 19).

SUMMARY

The etiological factors concerned in paralytic scoliosis are complex. Four main types of paralytic scoliosis can be recognised.

1. The general C-curve due to the body's anatomical attempt to shift its centre of gravity towards the weaker side. Vertebral rotation is not usually marked. This type usually occurs when patients with relatively slight paralysis have been allowed up too early; it does not usually progress to severe deformity but may occasionally do so, gradually changing into Type 2. This type usually responds well to a period of rest and muscle redevelopment in recumbency. It also responds favourably to correction and fusion because correction is easy and there is little tendency to deterioration. Many of the " successes " of correction and fusion are in this class—almost equal success would often have been gained without " correction." The spine is slightly, but not very, unstable and a relatively localised fusion will give the little extra support that is needed.
2. The " general collapse " type of curve due to extensive spinal weakness. This is the type in which simple head suspension produces marked correction. Rotation is moderate. Provided the patient's general condition is satisfactory extensive spinal fusion is usually the best treatment and produces gratifying improvement.
3. The primary lumbar curve due to a combination of pelvic obliquity, extraspinal imbalance and imbalance of the deep rotator muscles. Rotation is usually marked. Treatment must include the correction of all these factors. In mild cases correction of the pelvic obliquity is enough, but in marked cases the spine must also be corrected. The disability from a lumbar paralytic scoliosis is much greater than that from a lumbar idiopathic scoliosis of the same degree; so correction is necessary in this type. Correction in a Risser-type jacket is often inadequate and recourse to operative correction is usually required.
4. The primary thoracic curve—often associated with weakness of the scapular muscles. The indications for and methods of treatment are practically the same as in primary idiopathic thoracic curves. These curves tend to be progressive and uncompensated. Although the most popular treatment is correction and fusion, wedge osteotomy of the spine gives better correction in intractable cases.

The main need is for further investigation into the etiology of paralytic scoliosis so that adequate preventive measures may be undertaken at an early stage. It is essential that every child who contracts poliomyelitis should have his back muscles examined before he gets up. If there is any suggestion of scoliosis further investigations including radiography and electromyography are essential.

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PARALYTIC SCOLIOSIS

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A study of the sparse literature on paralytic scoliosis shows how little we know of this deformity. The differentiation of paralytic scoliosis into patterns, and the prognosis, are hardly mentioned. It seems that this deformity, like those seen elsewhere after poliomyelitis, is produced by unequal muscle pull. This has more often been stated than proved. Colonna and Vom Saal (1941) reviewed 500 cases of poliomyelitis including 150 with paralytic scoliosis. They attempted to correlate muscle paralysis with the type of curvature that developed. The evidence to be presented in this contribution will, it is believed, show that most of their conclusions are incorrect although some are confirmed. I would however like to pay tribute to these authors as the originators of important principles relating to the development of paralytic scoliosis.

A series of 193 patients with paralytic scoliosis has been studied. The aim was to classify the scolioses into groups based on their anatomical site, to discover the prognosis, and to correlate the associated muscle paralysis with each. In addition, a group of 280 patients with poliomyelitis but without scoliosis has been studied. Children with an average of seven years from the onset of disease showed frequent asymmetrical paralysis of limb muscles but no curvature of the spine.

In idiopathic scoliosis the division into groups based on the anatomical site of the primary curve has considerably advanced our understanding. A rather similar group of curve patterns exists in paralytic scoliosis. However, these groupings are not clear-cut, nor is each group homogeneous within itself. This is not surprising because the degree of muscle imbalance and the age at which paralysis occurred are more important prognostic features than the anatomical situation of the primary curve, whereas in idiopathic scoliosis the site of the primary curve is the main determinant of prognosis.

It is not difficult to decide which of the curves is the primary one. The distinction between a primary and compensatory curve is largely clinical, and relies on the fact that fixed structural rotation persisting on forward flexion occurs only in primary curves—this being more strictly true of paralytic than of idiopathic curves.

Arm and leg muscles that might be related to scoliosis have been charted, using the Medical Research Council grading 0–5. It would appear that paralysis of these muscles is not related to the development of scoliosis. Colonna and Vom Saal recorded only the number of occasions on which certain muscles were found to be paralysed in relation to the concavity of a curve—for example the trapezius in thoracic curves. In this series limb muscles paralysed on both sides were studied, and the numbers were found to be approximately equal.

In the trunk a relationship between muscle weakness and scoliosis has been found, but not that which has commonly been accepted. In brief, weakness of the mid-line muscles such as the erector spinae and the anterior abdominals cannot be shown to have any statistical relationship to the development of lateral curvature, though they are related to antero-posterior deformities. Symmetrical weakness of these muscles causes a collapse of the spine and thus a non-structural lateral curve. It is highly probable that this collapse is in part the cause of structural lateral curvatures of the spine.

The lateral trunk muscles—the intercostals, lateral abdominal muscles and to a small extent the latissimus dorsi—do however bear a very interesting relationship, being weak on the convexity in a highly significant number of patients. These muscles act on the spine through the long levers of the ribs and it would appear that this is an important factor.

These conclusions must be studied with the reservation that paralysis of the trunk muscles cannot be graded exactly. To reduce this source of error I have so far as possible done the charting myself. Fortunately, it is the asymmetry of right and left muscles that is important and this is often easy to determine, though the quantitative interpretation in terms of the Medical Research Council grading is not always reliable even with one observer. As this study is largely dependent on accurate muscle charting, a brief account will be given of the method as applied to the muscles which present special difficulty in assessment—the intercostals, erector spinae and the anterior and lateral abdominals.

Intercostal muscles—This study of the intercostal muscles was instigated by the development of a high thoracic curve in a patient who was known to have had poliomyelitis. This is the only cause of this type of curvature except congenital lesions and yet careful muscle charting revealed no abnormality. On the third study of this patient's muscles it was found that the apex of the chest on the side of the curve did not expand at all, in marked contrast to the other side.

The strength of the intercostal muscles is now measured at three levels. At the apices both hands are placed palm down, and their upward and forward excursions are compared. At the nipple line and the bases the fingers encircle each side as completely as possible while the thumbs touch one another anteriorly; the degree of excursion of each thumb on inspiration is noted. The correlation of the excursion with the Medical Research Council grading is most inexact and personal, but asymmetrical expansion is seen and felt quite easily and is found consistently; one hand may remain still while the other moves from one to one and a half inches. Of course, the accessory muscles of respiration must be relaxed.

Erector spinae muscles—It is always difficult or even impossible to detect minor localised weakness of the erector spinae muscles on one side as compared with the other, and this is especially so in scoliosis. Thus chartings here record the performance of both erectores spinae together and in their whole length. Study of the erector spinae over the length of the primary curve at operation often failed to reveal diminution or pallor of either muscle.

Anterior abdominal muscles—Orthodox examination of the anterior abdominal muscles has presented little difficulty. The movements of the umbilicus (Beevor's sign) are a sensitive indicator of abdominal asymmetry. Vertical asymmetry is not recorded here.

Lateral abdominal flexor muscles—This group, comprising quadratus lumborum and the lateral part of the abdominals, is difficult to assess. The patient is told to elevate the pelvis on one side while the leg is pulled downwards. The measurement of strength of these muscles is aided by placing a hand between the iliac crest and the ribs which are approximated by true action of these muscles but not by trick action of latissimus dorsi, the erector spinae on the concavity, nor by wriggling movements using the gluteal muscles. Visually this trick movement is not easily detected. It is important to make sure that lateral active flexion is not prevented by lumbar stiffness.

CURVE PATTERNS IN PARALYTIC SCOLIOSIS

The following curve patterns are regarded as distinct; they are similar to, but not identical with, those of idiopathic scoliosis. The numbers refer to the cases available for study; they total 193 patients.

Curve patterns	
High thoracic	39
Thoracic	69
Thoraco-lumbar	47
Lumbar	17
Combined thoracic and lumbar	13
"Telescopic" spine	8

These patterns will be studied particularly in relation to the associated muscle paralyses and to the prognosis.

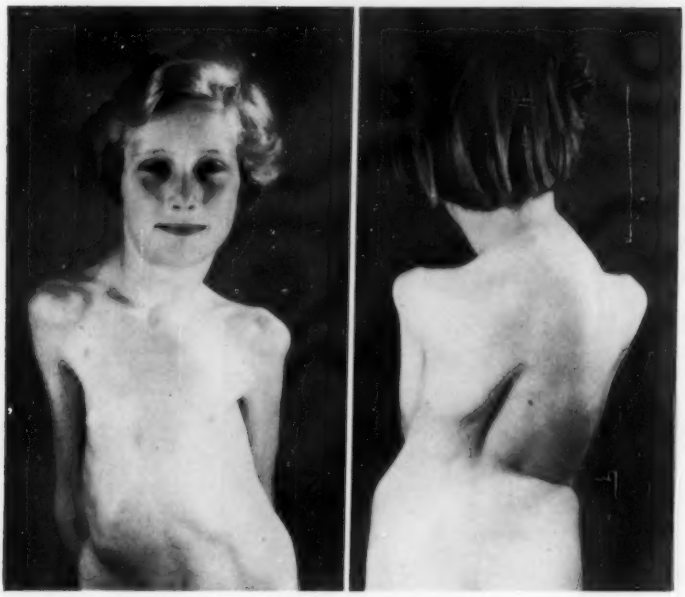

FIG. 1

High thoracic scoliosis. The right trapezius is elevated by the rotated upper ribs. The neck and head seem to be sidestepped on the trunk.

FIG. 2

High thoracic scoliosis with curvature of 120 degrees. The initial deformity is being disguised by the thoracic kyphosis and cervical lordosis.

HIGH THORACIC PARALYTIC SCOLIOSIS

I define this pattern as a primary curve in the upper thoracic region in which the highest vertebra of the primary curve is at the level of either the first thoracic (thirty-one cases) or second thoracic (seven cases). Only one curve extended into the cervical spine to the seventh cervical level.

This radiological definition is arbitrary, but early in this study it became apparent that there was a clinical type with an ominous prognosis and a most serious cosmetic effect. Rotation of the uppermost two ribs creates an elevation of the trapezius line of the neck. This is thrown into relief because the neck develops a compensatory curve in the opposite direction (Fig. 1). When the degree of curvature is great this may be disguised by the associated upper thoracic kyphosis, and by the compensatory cervical lordosis; the head then appears to be dropped between the shoulders (Fig. 2).

It is impossible to disguise this deformity by the dressmakers' art, unlike curves in other areas; even a collar and tie detracts little from the ugly deformity. It is to be remembered

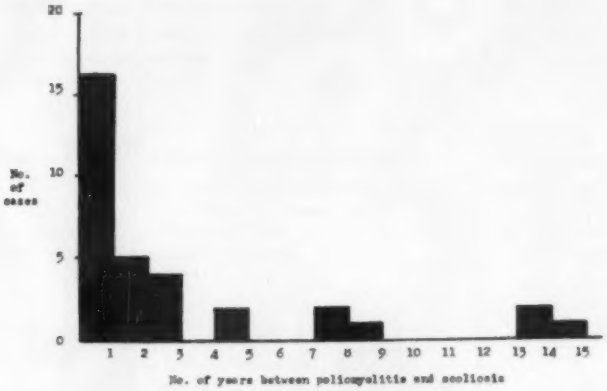

FIG. 3

High thoracic scoliosis. Interval between poliomyelitis and onset of scoliosis (thirty-three patients).

that the obvious deformity arises from rotation of the ribs and is therefore unaltered by surgical fusion, which therefore should be early and prophylactic, rather than curative. Knowledge of the prognosis in this curve is the basis of our therapeutic decision.

Thirty-nine patients suffering from high thoracic paralytic scoliosis were studied. There were twenty-two females and seventeen males. The primary curve was convex to the right in twenty-one and to the left in eighteen.

The age at which poliomyelitis occurred is, of course, scattered through the years of childhood. The oldest child to develop poliomyelitis and then scoliosis was a boy of fourteen years nine months, with a final high thoracic curve of 30 degrees. Structural scoliosis did not follow poliomyelitis in adult life. The interval between poliomyelitis and the onset of this pattern of curvature was recorded in thirty-three patients; it varied between a few weeks to fifteen years, but in half of them it was noted within one year (Fig. 3).

The curves starting at the first thoracic level extended variably between the sixth and eleventh thoracic vertebrae, and those beginning at the second thoracic from the ninth thoracic to second lumbar. Thus there might be from six to thirteen vertebrae in the primary curve. The distribution between these extremes was even. This length of curve is on the average greater than the idiopathic variety in which curves of twelve and thirteen vertebrae (four cases) are never seen.

Prognosis—In twenty of the thirty-nine patients the iliac apophyses had reached the posterior superior spine. Risser has shown that this is contemporaneous with the end of spinal growth, after which an idiopathic curve will not increase. In paralytic scoliosis such an end-point is not absolutely reliable but a noteworthy increase after this event was very rare in this series. The complete development in length of the iliac apophysis will therefore be taken to indicate the completion of curvature.

Figure 4 illustrates the final curvature of the twenty adult patients. How commonly gross deformity arises is well seen. Mild curves may be radiographically defined as those of less than 70 degrees (four cases), severe 70–99 degrees (one case), very severe 100 degrees or more (fifteen cases). The worst curve seen amounted to 155 degrees.

Among the remaining nineteen patients at present under observation who have not completed growth, there are four very severe curvatures, nine severe and six mild; four of the latter group are less than ten years of age. Eleven of these nineteen have already been operated upon and their natural evolution has been interrupted. The prognosis of the high thoracic paralytic curves untreated or conservatively treated is indeed gloomy.

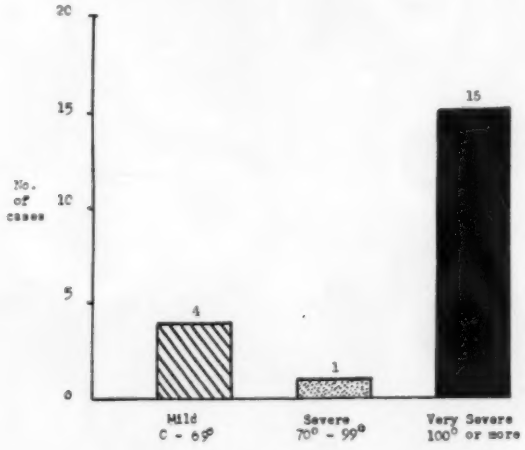

FIG. 4
High thoracic scoliosis. Twenty mature curves.

Colonna and Vom Saal came to the conclusion that " the upper extremity and the back muscles cause thoracic scoliosis." Their thesis was that the stronger arm muscles pulled the spine towards them. They investigated latissimus dorsi, trapezius and the rhomboids. They found latissimus dorsi to be paralysed thirty-four times out of sixty-four on the side of the concavity. The rhomboids they found to be weak on the concavity thirty-six times in forty-two cases and six times on the convexity, and in all twenty-eight cases of weak trapezius the affected side was the concavity of the curve. The deltoid was weak on the side of the convexity in twenty-three of forty-five cases. No statistics are given for the erector spinae. In their paper no information is available about the degree of muscle weakness present or of the existence of weak muscles on the convexity of the curve, and they did not discuss the existence of weak muscles without scoliosis.

In this series paralysis of the arm muscles is not thought to be significant; the commoner finding was weakness on the convexity, the opposite to that recorded by Colonna and Vom Saal. They did not mention the role of the intercostals, nor has any reference been made by any other author in a comparable manner. It is believed that it can be shown that the

intercostals on the convex side of the curve are the only muscles that are significantly paralysed. The intercostals exert through the ribs a very effective leverage in laterally flexing the spine. Before discussing the role of the intercostals it would be well to see the evidence on which this assertion is based.

Trunk muscles—In six patients the only weakness found was in the intercostals muscles; the curvatures in these cases were 127 degrees, 122 degrees, 100 degrees, 100 degrees, 81 degrees, 77 degrees; there is nothing here to suggest that other muscles play a part even in such severe curves.

Intercostals. High thoracic curves

Intercostal weakness towards the convexity	29
Intercostal weakness towards the concavity	0
Intercostals symmetrically weak (less than 2) or totally absent	5
Inadequate records	5
	39

Erector spinae. High thoracic curves

Erector spinae grade 4 to normal	32
Erector spinae less than 4	6
No record	1
	39

Lateral abdominal flexors. High thoracic curves

Lateral abdominal flexors. Symmetrical grade 4 to normal	26
Lateral abdominal flexors. Weakness on the convex side	4
Lateral abdominal flexors. Weakness on the concave side	5
No records or symmetrically weak less than 4	4
	39

Abdominal muscles. High thoracic curves

Abdominals grade 4–5, symmetrical	18
Abdominals less than grade 4, symmetrical (–ve Beevor)	16
Abdominals asymmetrical, weak towards convexity	2
Abdominals asymmetrical, weak towards concavity	2
No record	1
	39

Arm muscles—The muscles from the scapula to the trunk—trapezius, serratus anterior, rhomboids, pectoralis major and latissimus dorsi—have been charted. There is evidence to be referred to later which suggests that the scapulo-humeral muscles play no part.

In many cases the upper limbs have been normal and an attempt was first made to group the arm-trunk muscles as a unit.

High thoracic curves

Arm-trunk muscles. Normal both sides	15
Arm-trunk muscles. Weak on the convexity	10
Arm-trunk muscles. Weak on the concavity	6
Arm-trunk muscles. Symmetrically weak	8
	39

To judge the effect of individual muscles is more difficult because it is uncommon to find an isolated asymmetrical paralysis. The latissimus dorsi was particularly referred to by Colonna and Vom Saal, and grading was therefore recorded in detail.

	Cases
Latissimus dorsi grade 4 to normal, bilateral	20
Latissimus dorsi grade 4 weak on the convexity	7
Latissimus dorsi grade 4 weak on the concavity	6
Equally weak or no record	6
	39

An attempt was made with the other muscles to record the findings where the discrepancy on the two sides was notable between pairs of muscles irrespective of the other arm-trunk muscles.

	Patients
Trapezius weak on convexity	5
Trapezius weak on concavity	4
Serratus anterior weak on convexity	5
Serratus anterior weak on concavity	3
Pectoralis major weak on convexity	5
Pectoralis major weak on concavity	4
Rhomboids weak on convexity	8
Rhomboids weak on concavity	4

This slight predominance of weak arm muscles on the convexity is not statistically significant. It would, from the nature of the disease, be expected that in one-sided paralysis the anterior horn cells whose axons go to the arm and the intercostals of the same side might be involved more often and more seriously than on opposite sides.

If it is true that the arm-trunk muscles have no effect upon the spine, and scoliosis is seen only in the presence of intercostal paralysis, and arm paralysis when present is coincidental, then it should be possible to find a group of patients with arm paralyses but normal intercostal muscles and see what has happened to their spines.

There have fortunately been available the records of 280 patients who suffered from limb paralysis who have been followed over the last four to five years in a study of muscle recovery carried out by my colleague, W. J. Sharrard. From these records, and additional cases of my own, there is a group of thirty-eight patients who have had unilateral arm-trunk muscle involvement and retained normal power in the intercostals. Many of these patients developed poliomyelitis after injections into the arm. This usually results in severe paralysis of one arm with the trunk muscles unaffected. The follow-up period from the onset of poliomyelitis is in no case less than four years and the average is seven years. Although many of these patients are still not fully grown it is apparent from Table I and Table III that well over half of them should have developed thoracic scoliosis within the past seven years, or even four years. In fact, none has scoliosis. One patient had his spinal accessory nerve divided at the age of three years. Despite a total paralysis of one trapezius he had no scoliosis when seen at the age of twenty-six years. No scoliosis has been seen among a large number of these patients who have had paralysis of the upper limb distal to the arm-trunk muscles if the intercostals were normal.

Case 1—A boy of twelve years developed poliomyelitis at the age of four years, and scoliosis was noticed six months later. At the age of eleven years he had a curvature of 81 degrees to the left, extending from the first to the eighth thoracic vertebrae, with slight apical rib collapse (Figs. 5 and 6).

	R.	L.
	4	2
This boy had an intercostal paralysis:	4	1. In addition he had a weak right deltoid.
	4	4

Case 2—A girl aged fourteen years who developed poliomyelitis at the age of seven and scoliosis at eight years. There was paralysis of the right arm which has entirely recovered. After complete

FIG. 5
Case 1—Early high thoracic paralytic scoliosis. Note elevated left trapezius.

FIG. 6
Case 1—The radiograph shows a left high thoracic curve of 81 degrees, extending from T.1–8, with moderate rib collapse.

muscle charting no abnormality was found except in the intercostal muscles where the right side was much weaker. The charting was: R. 0 3, L. 0 3. Her vital capacity was 650 cubic centimetres. When she first attended she had a curve of 122 degrees with very severe collapse of the first to the tenth ribs on the right side (Figs. 7 and 8). Recent correction in a distraction jacket gave correction to 70 degrees.

FIG. 7

FIG. 8

Case 2—Characteristic high thoracic scoliosis. The radiograph (Fig. 8) shows a right high thoracic curve of 122 degrees, associated with intercostal paralysis and collapse of ribs. The primary curve extended from T.1–10.

THORACIC PARALYTIC SCOLIOSIS

In thoracic paralytic scoliosis the upper limit of the primary curve is at the level of the third thoracic vertebra or lower, but the apex of the primary curve is higher than the eleventh thoracic where the thoraco-lumbar pattern begins. In idiopathic scoliosis this group is the most common and most severe, but the remarkable restriction of severe curves to the thoracic region is not seen in paralytic curves. In this group of sixty-nine patients there were thirty-six females and thirty-three males. An unexpected and unexplained finding is that, as with idiopathic thoracic curves, there is a predominance of curves to the right—fifty-four to the right and only fifteen to the left. Colonna and Vom Saal found the distribution approximately equal. In this group of thoracic curves, though not in the high thoracic or other groups, the muscle paralysis has most often been right-sided. The great majority of these patients have developed thoracic scoliosis within two years of the paralysis. Figure 9 shows the interval between poliomyelitis and the discovery of scoliosis. Thoracic curves varied in site and length; they may extend as low as the fourth lumbar vertebra. The length of the primary curve varied between four to thirteen vertebrae, most being eight to ten vertebrae in length.

Prognosis—Thirty patients had completed spinal growth as indicated by the iliac apophysis. The prognosis of the thoracic group was, in the small series available, distinctly better than the high thoracic. Among the thirty mature curves eight remained mild, ten were severe

and twelve increased to more than 100 degrees (Fig. 10). Among the remaining thirty-nine immature curves seventeen were mild, thirteen severe and nine very severe; several patients had undergone fusion and their expected deterioration had been arrested.

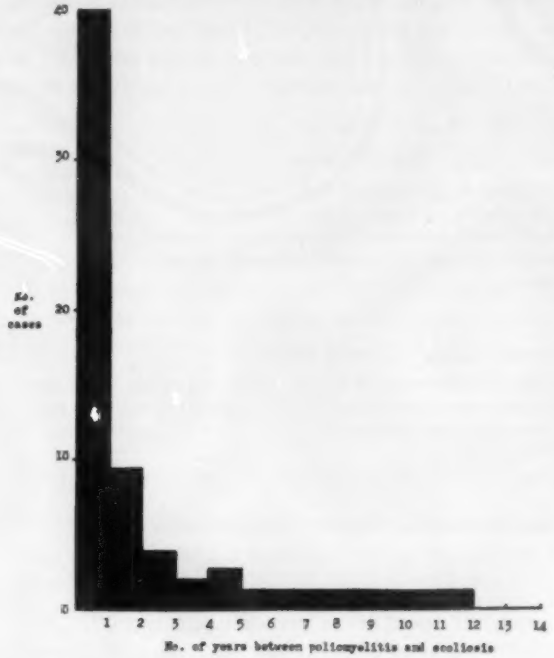

FIG. 9

Thoracic paralytic scoliosis. Interval between poliomyelitis and onset of scoliosis (sixty-two patients).

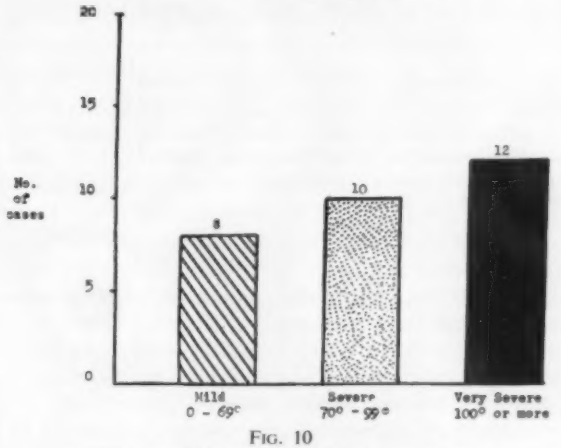

FIG. 10

Thoracic paralytic scoliosis. Thirty mature curves.

Muscle paralysis in relation to thoracic scoliosis—This group of scolioses has not so clear nor so consistent a relationship as the high thoracic curves. It might be expected that both arm-trunk and abdominal muscles might play their part, and play it differently, depending

on whether the curve starts at the third thoracic level or much lower. It is apparent, though less clear, that the intercostal muscles are, irrespective of level, still the dominant cause of deformity.

Trunk muscles—Six patients with normal or good intercostal muscles had rather small curves—86 degrees, 60 degrees, 50 degrees, 49 degrees, 30 degrees and 23 degrees respectively. All but one of these patients showed no rib collapse, and one showed only slight collapse. The cause of the scoliosis in these patients was not apparent. Two patients had paralysis limited to the intercostal muscles.

Intercostals. Thoracic curves	
Intercostals symmetrical 4–5	6
Intercostals symmetrical less than 4	2
Intercostals asymmetrically weak on convexity	41
Intercostals asymmetrically weak on concavity	1
Inadequate or no records	19
	69

Erector spinae. Thoracic curves	
Erector spinae normal 5	35
Erector spinae grade 4	17
Erector spinae less than 4	15
No record	2
	69

Lateral abdominal flexors. Thoracic curves	
Lateral abdominal flexors grade 4 to normal	33
Lateral abdominal flexors symmetrical less than 4	10
Lateral abdominal flexors weak on convexity	15
Lateral abdominal flexors weak on concavity	8
No record	3
	69

Ten of these with weak lateral flexors on the convexity had primary curves involving lumbar vertebrae.

Anterior abdominal muscles. Thoracic curves	
Anterior abdominal muscles 4 to normal, symmetrical	9
Anterior abdominal muscles symmetrical, less than 4	48
Anterior abdominal muscles weak on convexity	8
Anterior abdominal muscles weak on concavity	3
No record	1
	69

Of the eight patients with weak abdominals on the convexity, seven have primary curves including the first to the fourth lumbar vertebrae.

Arm muscles	
Arm-trunk muscles. Thoracic curves	
Arm-trunk muscles normal both sides	39
Arm-trunk muscles equally weak	13
Arm-trunk muscles weak on convexity	9
Arm-trunk muscles weak on concavity	7
No record	1
	69

Latissimus dorsi. Thoracic curves	
Latissimus dorsi grade 4 to normal	40
Latissimus dorsi equally weak right and left	6
Latissimus dorsi weak on convexity	13
Latissimus dorsi weak on concavity	5
No record	5
	69

Trapezius weak on convexity	2	Serratus anterior weak on convexity	4
Trapezius weak on concavity	4	Serratus anterior weak on concavity	3
Pectoralis major weak on convexity	5	Rhomboids weak on convexity	4
Pectoralis major weak on concavity	5	Rhomboids weak on concavity	2

A number of these patients wore abduction frames for paralysis of one upper limb. This has been said to be a factor in producing scoliosis. Among the 280 patients previously mentioned there were ninety-seven who wore an abduction frame for more than four months and none of these had developed curvature when last seen.

Case 3—A girl aged eleven years developed poliomyelitis. She now has a curve of 120 degrees (right) extending from the fourth to eleventh thoracic vertebrae. She was not seen until the age of fifteen years when surgical fusion was undertaken. All arm muscles are grade 4. Intercostals:

	R.	L.
	1	3
	1	3.
	0	3

The ribs are collapsed in the mid-zone from about the third to the eighth ribs (Figs. 11 and 12).

Case 4—An Anglo-Indian boy developed poliomyelitis at the age of one year and now at the age of ten years his curve measures 96 degrees. This proved correctable to 29 degrees and fusion was carried out. His vital capacity was 600 cubic centimetres. Intercostal charting was:

	R.	L.
	3	3
	1	3.
	0	3

The erector spinae muscles were normal. The left arm (on the side of concavity) was weaker than the right (Figs. 13 and 14).

The role of the intercostals in thoracic scoliosis—In high thoracic paralytic scoliosis the intercostals seem to be the only muscles consistently paralysed on the convex side of the curve. In thoracic scoliosis they are more commonly paralysed on the convex side than any other muscle, but there are six patients who have normal intercostal muscles and scoliosis. The anterior abdominal muscles and the lateral abdominal flexors appear to play a part, and it is noteworthy that in the cases in which they may be deforming, the curvature is at the low thoracic level and involving the lumbar vertebrae—in fact they resemble thoraco-lumbar curves. The arm and trunk muscles seem to play no part, and it is to be noted that charting of these muscles is very accurate in comparison to the trunk muscles.

The marked asymmetry of the intercostal muscles as measured by chest expansion is often localised to the area of the curve. This could be secondary to chest deformation from the scoliosis rather than from paralysis of the intercostals. However, during the period of observation of these patients several hundred patients with idiopathic scoliosis have been examined by muscle charting, and asymmetric intercostal movement had not been found in scoliosis from this or other causes. Because the ribs are crowded together on the concave side of the curve it would seem that if there was any restriction it should be found on this side.

An important aspect of intercostal paralysis which has seldom before been more than briefly mentioned is the phenomenon of rib descent. On the convex side of the curve, and usually limited to the area of the thoracic curvature, the ribs are strikingly deformed and crowded. The main deformity is a rib droop. Instead of being horizontal they become almost vertical, and sometimes completely so. These vertically lying ribs are crowded together and

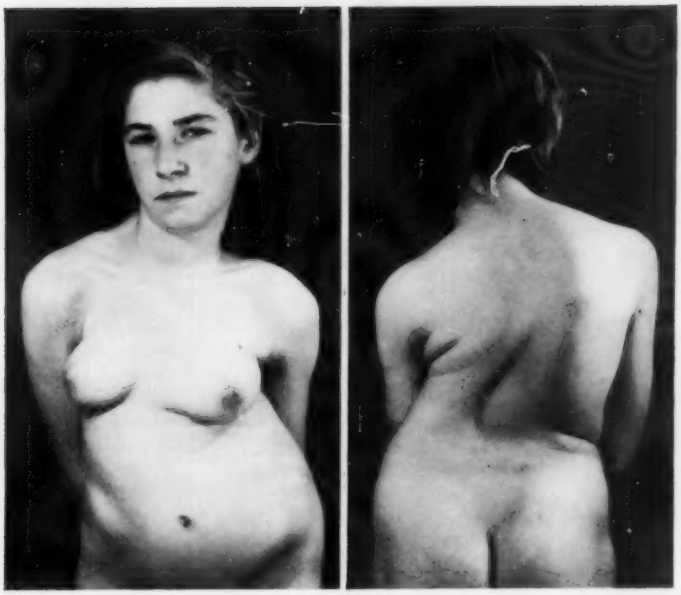

FIG. 11
Case 3—Thoracic paralytic scoliosis. Right thoracic curve of 120 degrees.

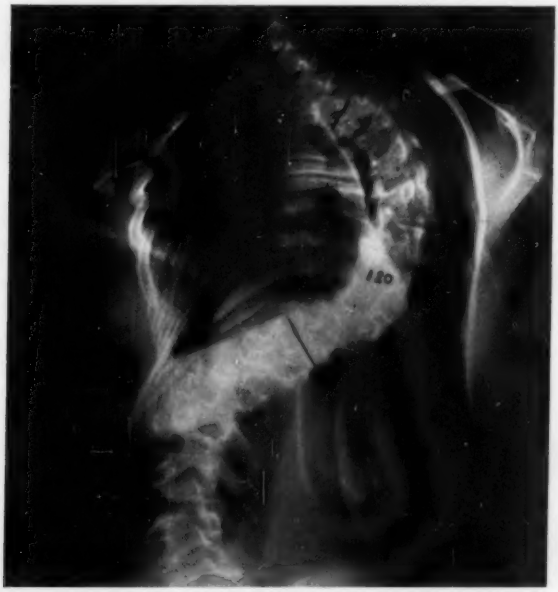

FIG. 12
Case 3—A curve of 120 degrees between T.4–11. Middle ribs 3–8 are collapsed. Iliac apophyses almost complete.

FIG. 13
Case 4—Before operation.

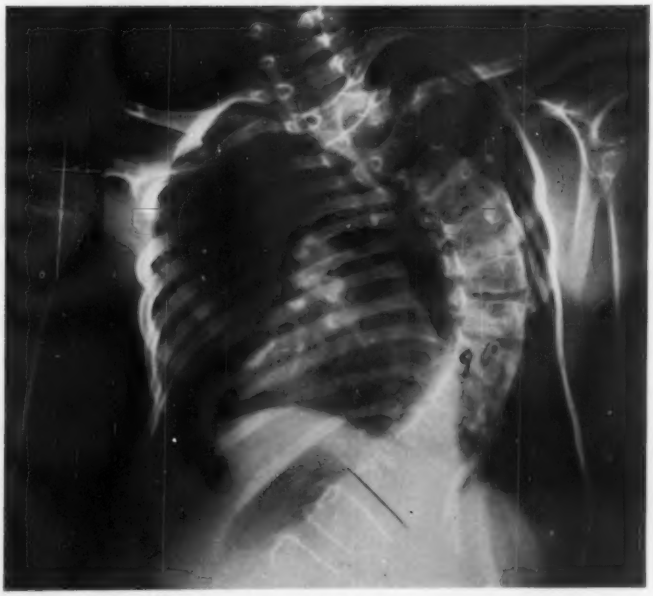

FIG. 14
Case 4—A typical thoracic curve of 96 degrees between T.5 and L.1. Mid-zone rib collapse. Weak intercostals.

lie one behind the other. Their position is the opposite of that of the ribs in idiopathic scoliosis, in which the ribs on the convex side of the curvature may even spread apart, and it is the ribs on the concave side which crowd together; there may be some change from the horizontal towards the vertical but only comparable to that seen in slighter cases of paralytic rib drooping.

Among the thirty-nine high thoracic curves all showed structural changes in the ribs, and in only three could it be called slight. Among the sixty-nine thoracic curves there were fifty-eight with abnormal rib position. The eleven with normal ribs included the six patients without detectable paralysis of intercostal muscles. One patient, with a left thoraco-lumbar scoliosis, has a right apical rib collapse and no scoliosis. Otherwise rib change without thoracic scoliosis has not been seen in children.

In an attempt to confirm the view that these rib changes were indicative of intercostal paralysis, ten patients were observed by cineradiography. In each of these patients one respiratory cycle was screened and photographed. This strip of film was joined to make a continuous film which can be projected indefinitely from a special projector so that one respiratory cycle can be studied for as long as desired.

In brief, the results of this study of rib movements showed that these vertical ribs ascended through about half the excursion of the rib on the other side. It was often clearly seen that rib movements, which were small in the ribs attached to the vertebrae of the primary curve, were normal or greater than normal in the remaining ribs of the same side. Clinically the rib droop is associated with a razor-edge rotation; and radiographic evidence of rib deformity, or the clinical finding of razor-edge deformity, is most suggestive of paralytic scoliosis. Figures 15 to 18 show these rib changes.

Illustrative Cases

Case 5—This girl, now aged twenty-seven years, had poliomyelitis at the age of nine years and developed a high thoracic curve of 121 degrees. Intercostal charting is recorded as:

	R.	L.
	2	0
	2	0
	3	3

Figure 15 shows the first to the seventh ribs to be dropped. The upper ones are almost vertical.

Case 6—A girl now aged nineteen years developed poliomyelitis at the age of thirteen years. Despite this late onset she developed a severe high thoracic scoliosis of 125 degrees (Fig. 16). The first to the seventh ribs on the convex side of the spinal curve are almost vertical, lying one behind the other.

Case 7—This girl aged eleven years died after atelectasis—a recurrent event since she had been in a respirator for three months at the onset of paralysis eight years before. The two radiographs taken with an interval of three years are seen in Figures 17 and 18. They show characteristic early paralytic rib collapse which later became fully developed.

THORACO-LUMBAR PARALYTIC SCOLIOSIS

This group, intermediate between the well defined thoracic and lumbar curves, is a mixture of the two. The curves themselves are variable, and relating the paralysis to the curvature is on many occasions difficult or impossible. Restriction of the term thoraco-lumbar to those curves in which the eleventh or twelfth thoracic vertebrae form the apex follows the definition accepted in idiopathic thoraco-lumbar scoliosis.

Forty-seven cases have been studied, of which twenty-five were in girls and twenty-two in boys. There were thirty-four curves to the right and thirteen to the left. Although less closely related to muscle paralysis in this area, the large number of right-sided curves is as difficult of explanation as in the thoracic region. The unknown factor by reason of which 80 to 90 per cent of idiopathic thoracic curves are right-sided may well be operating here. The frequent occurrence of scoliosis within two years of the onset of paralysis is demonstrated in Figure 19.

The site of these curves varies considerably and they may involve from six to thirteen vertebrae, usually one or two more than their idiopathic counterparts.

FIG. 15

FIG. 16

Figure 15. Case 5—High thoracic curve of 121 degrees. Collapse of ribs 1–7. There is no chest expansion in the upper two-thirds of the left chest and the ribs are seen to be drooped and crowded. Figure 16. Case 6—Collapse of ribs 1–7.

FIG. 17

FIG. 18

Case 7. Figure 17—Scoliosis of 58 degrees and early rib collapse. Figure 18—Two and a half years later curvature has increased to 118 degrees and the rib collapse now resembles that seen in nearly all cases of high thoracic scoliosis.

Rib collapse, almost always basal, was seen in twenty-five patients; seventeen had none; and in four no record was available. Only eighteen of this group had completed growth. It is a small number from which to obtain significant facts, but the prognosis of these is

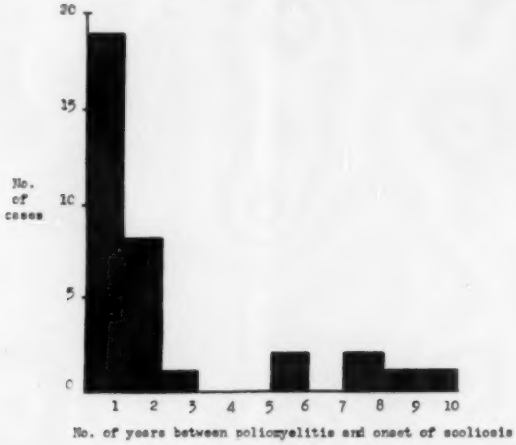

FIG. 19
Thoraco-lumbar paralytic scoliosis. Interval between poliomyelitis and onset of scoliosis (forty-seven patients).

represented in Figure 20. The remaining twenty-nine patients, who are still growing, showed twelve mild curves, eleven severe and six very severe; some have been operated upon.

Muscle paralysis in thoraco-lumbar scoliosis—Difficulty is found in explaining some of these in terms of asymmetrical muscle paralysis. In this grouping also we find an important number

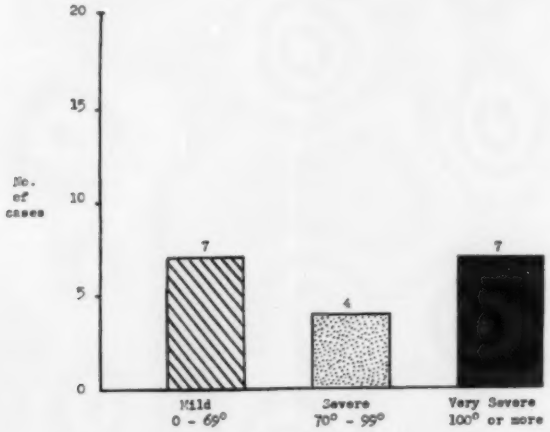

FIG. 20
Thoraco-lumbar paralytic scoliosis. Eighteen mature curves.

of curvatures related to a symmetrical weakness of muscles; a " telescopic " spine appears slowly to assume a structural curve from gravity rather than muscle pull. This will be discussed again. The trunk muscles will first be reviewed.

Trunk muscles

Erector spinae. Thoraco-lumbar curves

Erector spinae symmetrical 4 to normal	20
Erector spinae symmetrical less than 4	26
No record	1
	47

Lateral abdominal flexors. Thoraco-lumbar curves

Lateral abdominal flexors symmetrical 4 to normal	15
Lateral abdominal flexors weak on convexity	21
Lateral abdominal flexors weak on concavity	2
Lateral abdominal flexors less than grade 4 symmetrical	8
No record	1
	47

Anterior abdominal muscles. Thoraco-lumbar curves

Anterior abdominal muscles grade 4 to normal symmetrical (–ve Beevor)	6
Anterior abdominal muscles weak on convexity	8
Anterior abdominal muscles weak on concavity	2
Anterior abdominal muscles symmetrically weak	31
	47

Intercostals. Thoraco-lumbar curves

Intercostals symmetrical 4 to normal	10
Intercostals weak on convexity	11
Intercostals weak on concavity	4
Intercostals symmetrically weak less than 4	8
No record	14
	47

The arm muscles, gluteus maximus, psoas and leg abductors were checked and were not related. The noteworthy feature in these muscle charts was the frequency with which the lateral abdominal flexors were weak on the convexity. This will be discussed more fully in the lumbar group. The intercostals may play a small part; basal weakness was found on the convexity on a number of occasions. The other aspect of muscle weakness was the frequency of weakness of the erector spinae (twenty-six cases) and of the anterior abdominal muscles (thirty-one cases). This might be found in the presence of symmetrically strong lateral flexors, with marked lateral asymmetry, or all three groups might be equally weak on the right and left sides. In two instances almost no paralysis could be detected and in one other only the anterior abdominals. Although I believe that collapse of the spine from the force of gravity is important in the etiology of thoraco-lumbar scoliosis it should be made clear that the cause of this curvature in terms of precise muscle paralysis is not explained in a number of cases.

A real shortening of one leg of more than one inch occurred on the convexity (where it might be expected) three times, on the concavity five times. Contracture of tensor fasciae latae, said by Irwin (1949) to be a cause of paralytic scoliosis, occurred bilaterally once, on the side of the convexity three times, and on the concavity once. Pelvic obliquity, a feature of paralytic scoliosis in the lumbar region, occurred eleven times, in all but one in association with inequality of the lateral flexors, this inequality being the cause of pelvic obliquity (Mayer 1936).

Illustrative Case

Case 8—Now fourteen years old, this patient had developed poliomyelitis at the age of seven years. One year afterwards curvature was noticed and progressed rapidly. At the age of ten a fascial graft was placed between the left iliac crest and ribs to try to prevent further deformity arising from the totally paralysed lateral flexors and intercostals on the left side. It did not slow deterioration and the curvature increased to 130 degrees and extended over eleven vertebrae from the fifth thoracic to the

third lumbar, the apex being at the eleventh and twelfth thoracic. She had a gross pelvic obliquity from the inequality of the lateral flexors, right 4, left 0. The intercostal charting was:

R.	L.
2	2
1	1.
1	0

Gross muscle imbalance accounted for this curvature, but in addition the abdominals and erector spinae were symmetrically weak and allowed additional collapse (Figs. 21 and 22).

FIG. 21

FIG. 22

Case 8. Figure 21—Thoraco-lumbar scoliosis. Gross inequality of the lateral abdominal flexors together with collapse due to symmetrical weakness of other muscles caused this severe thoraco-lumbar curve. Figure 22—A long 130 degrees thoraco-lumbar curve with rib collapse and marked pelvic obliquity.

LUMBAR PARALYTIC SCOLIOSIS

This group of cases, few in number, provides a clear cut entity with a related muscle paralysis, as noted by Colonna and Vom Saal. There were seventeen cases of lumbar paralytic scoliosis, eleven male and six female. Twelve were right sided, four left. The interval between the paralysis and the onset of curvature is on the average longer than in the other groups. Only twelve patients had completed growth. These numbers are too small to be significant. However, the number of mild cases was two, severe seven and very severe three. It would appear that the prognosis is little better than in other groups—very different from the idiopathic curves of this region.

The top vertebrae of these curves varied between the levels of the sixth thoracic to the first lumbar. The apex, however, was always in the lumbar vertebrae. The length of the curve varied between five and thirteen vertebrae.

The important group of muscles causing lumbar scoliosis appears to be the lateral abdominal flexors—the quadratus lumborum, lateral portion of the anterior abdominal muscles, and in two patients perhaps the latissimus dorsi. In established curves, trick elevation of the pelvis can be produced by the erector spinae but it is never powerful.

Associated with imbalance between the lateral flexors is a pelvic tilt. The strong lateral flexors pull the pelvis up on the side opposite the convexity. Apparent shortening must

accompany this and may be as much as three inches. Early in this pelvic elevation the soft-tissue contracture becomes fixed and the pelvic tilt cannot be altered by manual pull on the leg. In these seventeen patients there was a notable pelvic obliquity in twelve, and imbalance between the lateral flexors in the same twelve. One had no abnormal muscles except his right lateral flexor group which was grade 2. He developed one and a half inches of apparent shortening and an 88-degree curvature to the right (Figs. 23 and 24).

The remaining five patients with normal or symmetrically weak lateral flexors had considerable generalised trunk weakness and are regarded as having " telescopic " spines.

The detailed muscle charting proved to be as follows:

Lumbar paralytic scoliosis	
Erector spinae 4 to normal . . .	6
Less than 4 symmetrical	11
	17
Lateral abdominal flexors grade 4 to normal	3
Symmetrically weak less than 4 . .	2
Asymmetrically weak on the convexity .	12
Asymmetrically weak on the concavity .	0
	17
Anterior abdominals grade 4 to normal .	3
Symmetrically weak less than 4 . .	11
Asymmetrically weak on convexity . .	2
Asymmetrically weak on concavity . .	1
	17

It seems that if the intercostals play any part at all it is only small. Gluteus maximus, psoas and the leg abductors were in no way related. In three patients there was more than one inch of true leg shortening on the convex side, in two on the concave side. Three patients had slight bilateral contractures of the tensor fasciae latae.

It would seem from this series that, except when due to spinal collapse, lumbar paralytic scoliosis (always associated with pelvic obliquity and apparent shortening) is due to asymmetrical weakness of the lateral abdominal flexors. These are laterally placed muscles like the intercostals acting through long levers.

Illustrative Cases

Case 9—A boy aged twelve who developed poliomyelitis at five years of age and scoliosis at seven years. The only persisting paralysis was of the right lateral flexors, grade 2. The left were normal. He developed a lumbar curve from the tenth thoracic to the fourth lumbar vertebra of 88 degrees. There was one and a half inches of apparent shortening of the left leg and pelvic obliquity. His deformity is typical of most lumbar curves from lateral flexor imbalance (Figs. 23 and 24).

Case 10—In contrast, this man aged twenty years has a " collapse " type of lumbar curve. Although he developed poliomyelitis at the age of four years his curve was not noted until he was fifteen years of age. It is very long, involving thirteen vertebrae, and is only of 70 degrees, two typical features of collapse curves. He has symmetrically weak erector spinae and abdomen, but he did have slight asymmetry of the lateral flexors (Figs. 25 and 26).

One-sided absence of gluteus maximus may produce a non-structural curve when sitting, due to a unilaterally wasted buttock; this can be corrected by a block under the buttock. Muscle paralysis in this region of the body has also been investigated in the same large group of 280 patients previously referred to in discussing arm paralysis in relation to thoracic scoliosis. Among the children in this group inequality of the psoas muscles of more than one

FIG. 23 FIG. 24

Case 9. Figure 23—An isolated right lateral abdominal flexor weakness and 88 degrees of lumbar scoliosis. Figure 24—Lumbar curve with gross obliquity of pelvis. The approximation of ribs to pelvis by the stronger lateral flexor is well illustrated.

FIG. 25 FIG. 26

Case 10—Lumbar paralytic scoliosis. Figure 25—Lumbar "collapse" curve. Figure 26—Radiograph showing the long curve. Some pelvic obliquity.

grade occurred twenty-two times but no scoliosis developed. Similarly twenty-one asymmetrically paralysed glutei maximi were noted without curvature. In this same group of paralysed children the lateral flexors were symmetrically paralysed fourteen times and in one instance only was asymmetry present without scoliosis, lateral flexors of grade 3 : 0 being found in a boy of six without curvature. However, in some of the thoracic scoliotics reviewed there has been abdominal lateral flexor imbalance which has not produced scoliosis in the lumbar region.

Irwin is of the opinion that one tight tensor fasciae latae is a cause of scoliosis. No instance of scoliosis and a tight tensor fasciae latae has been found in which there was not also adequate lateral flexor paralysis to explain the deformity. Amongst forty-seven thoraco-lumbar curves unilateral tightness of the tensor fasciae latae was found three times on the convexity, once on the concavity. In seventeen lumbar curves no instance of unilateral tightness of tensor fasciae latae was found. Twelve cases of unilateral contracture have been observed among the 280 patients; none developed scoliosis. Apart from the mild non-structural compensatory scoliosis from the hip abductor contracture it is believed that this deformity is not significant. Shortness of a leg has in this series borne no relationship to the curvature. In a special clinic for leg inequality no structural scoliosis has been seen, nor have unequal legs been found to be relevant in a series of several hundred idiopathic scoliotics.

COMBINED THORACIC AND LUMBAR PARALYTIC SCOLIOSIS

The last and smallest group (fifteen cases) need not be discussed in detail. However, it does exist and it is important that it should be recognised because fusion of only one primary curve may be followed by an increase in the second primary curve with disastrous result. The apophyses of the ilium had completed their growth in only six cases. In four, both primary curves remained mild; the other two were respectively 116 degrees : 79 degrees and 150 degrees : 104 degrees. The relationship of these curves to the muscle paralysis was often puzzling. However, in eight instances the intercostal muscles were weak on the side of convexity of the upper primary curve, and never on the concave side of the deformity. Three patients had normal intercostals and two were not recorded. In five patients weakness of the lateral flexors occurred on the convexity of the lower curve, in four on the concavity but two of these had both curves confined to thoracic vertebrae. All other relevant muscles were charted and found not to be significantly related to the curvature.

Case 11—A girl aged ten developed poliomyelitis and this was immediately followed by scoliosis. She now has a right thoracic curve of 78 degrees from the third to tenth thoracic vertebrae and a left lower primary of 70 degrees from the eleventh thoracic to fourth lumbar. There was slight weakness of the right intercostals but no other evident related weakness (Figs. 27 and 28).

In idiopathic scoliosis the double primary pattern is common and uniform in character. In paralytic scoliosis it is uncommon and variable and believed to be due to a fortuitous paralysis of deforming muscles on opposite sides in the upper and lower curves respectively.

TELESCOPIC SPINE

In paralytic scoliosis there is often a considerable difference in the erect and supine curve measurements. This is the natural result of weakness of the trunk muscles and difficulty in overcoming the effect of gravity. The curve in a collapsing spine will usually diminish when the patient lies down, or more remarkably, in suspension. The importance of this factor in thoraco-lumbar and lumbar curves has already been stressed; it should always be remembered.

There were, however, in this group, eight patients who had gross but symmetrical anterior, lateral abdominal and erector spinae paralysis rarely above grade 3. These patients did not develop severe structural lateral curves although they did tend to produce correctable and

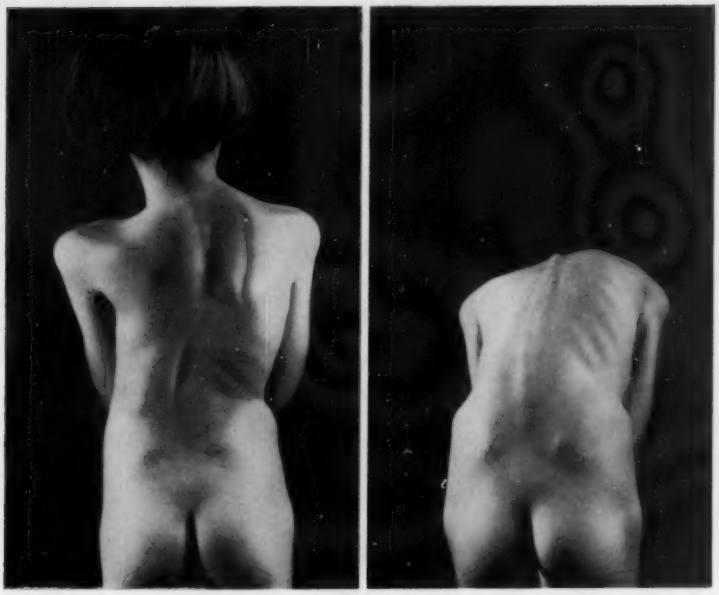

FIG. 27

Case 11—Combined thoracic and lumbar paralytic scoliosis. The right thoracic rotation is evident in both photographs but lumbar rotation is difficult to see except in the erect view.

FIG. 28 FIG. 29

Figure 28. Case 11—Two primary curves, 78 degrees and 70 degrees. Figure 29. Case 12—Antero-posterior deformities. Erector spinae graded 0/0/3 0/0/2. Abdomen 2. Lateral flexors 3.

non-structural thoraco-lumbar curves on sitting or standing. Many of these patients have flail spines and require long fusions for stabilisation.

In addition to the lateral curves developed, paralysis of the erector spinae and anterior abdominal muscles produces quite marked antero-posterior curves. Thoracic kyphosis with lumbar lordosis is seen in all eight patients (Fig. 29). It is believed that paralysis of the mid-line muscles produces antero-posterior and not lateral curves as its major effect, a view earlier expressed by Steindler.

An unusual type of paralysis has been seen on three occasions, a rather strictly segmental lumbar paralysis of erector spinae and the abdominals of the same segments. Gravity produces a small lumbar kyphosis in the erect position (Fig. 30).

FIG. 30
Localised erector spinae paralysis with kyphosis.

PROGNOSIS AND THE AGE OF ONSET

In idiopathic scoliosis the age at which curvature begins has an important bearing on prognosis, but less so than the site of the primary curve.

In paralytic scoliosis it has been demonstrated that the site of the primary curve is less important in prognosis than is the degree of muscle imbalance. Just as in the limbs, so in the trunk, muscle imbalance arising early in childhood causes a more severe deformity than does paralysis towards the end of growth. A total of ninety-two patients were found to have completed growth without alteration of their natural development by operation. In Figure 31 it can be seen

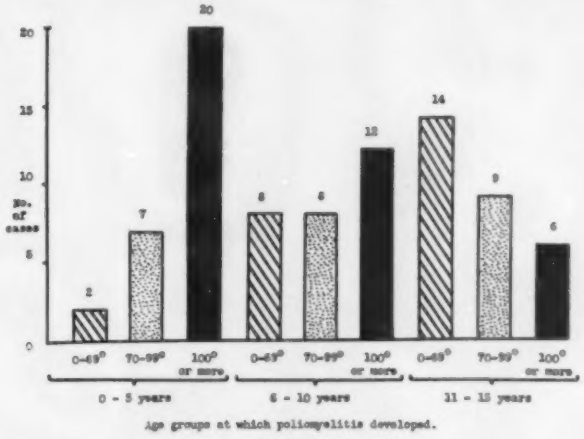

FIG. 31
Relationship of final curvature to age at onset of poliomyelitis.

that children who developed poliomyelitis before five years of age developed more serious deformities than did those in the two groups, six to ten years and eleven to fifteen years. In the youngest group the most common final curve is more than 100 degrees whereas in the oldest group the most common final curvature is less than 70 degrees.

POLIOMYELITIS IN ADULTS

Structural lateral deformity has not been seen in any girl who developed poliomyelitis after the age of fourteen or boy after fifteen. Thirty-four adults followed up over several years who had sufficient and appropriate paralysis for the development of scoliosis failed to deform.

THE RELATIONSHIP OF POLIOMYELITIS TO IDIOPATHIC SCOLIOSIS

It has often been said that idiopathic scoliosis is due to unrecognised poliomyelitis. There are many reasons why this cannot be, despite recent electromyographic evidence (Riddle and Roaf 1955).

Routine complete muscle charting of idiopathic scoliosis has not revealed isolated paralysis of limb muscles. The suggestion that several hundred children have been afflicted with isolated paralysis of their rotatory muscles leaving all other muscles unaffected and without a history of acute illness is too unlikely to be maintained. Moreover, in idiopathic scoliosis the curves are shorter and less mobile on the average, and do not present a high thoracic pattern. Finally, 90 per cent of patients with idiopathic scoliosis, appearing after ten years of age, are females.

SUMMARY

1. The prognosis of paralytic scoliosis has been studied by defining curve patterns and establishing the natural development as seen in fully grown patients who have not had surgical correction.

2. The prognosis, unlike that in idiopathic scoliosis, is related to the age of onset of the curvature and the degree of muscle imbalance rather than the site of the primary curve.

3. Paralysis of limb muscles is shown to be unrelated to the development of scoliosis. The intercostal muscles and the lateral abdominal flexors produce scoliosis when weaker on the convex side of the curve. Gravity and the other trunk muscles certainly play a part in the development of lumbar curves but their importance is difficult to assess.

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ARTERIAL DISEASE AS A CAUSE OF PAIN IN THE BUTTOCK AND THIGH

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When a patient attends an orthopaedic clinic with the complaint of pain affecting the calf of the leg, the possibility of arterial disease as a cause of the pain is usually remembered. When the pain is in the buttock or thigh, that possibility is often overlooked, although occlusion at the aortic bifurcation or in the iliac arteries can determine pain in such sites. Indeed, many of those who have studied the condition of thrombosis at the aortic bifurcation (Holden 1946, Elkin and Cooper 1949, Ortner and Griswold 1950, De Bakey *et al.* 1954, de Wolfe *et al.* 1954) believe that the condition is probably more common than is generally realised.

In the past four years (1952–55) nine patients have been seen at the Royal National Orthopaedic Hospital and one at St Mary's Hospital, in whom pain in the buttock or thigh was demonstrably caused by aortic or iliac occlusion. All, before being seen at these hospitals, had been considered to be suffering from conditions such as osteoarthritis of the hip or lumbo-sacral disc degeneration, and had received treatment accordingly. All these patients had escaped surgical treatment, but the author is informed by Professor C. G. Rob that he has seen a patient who underwent spinal fusion for pain from aortic thrombosis, and he himself has seen a patient who had had a laminectomy performed for pain in the calf due to a femoral thrombosis. Mistakes in the diagnosis of these cases may not only lead to mistaken treatment, but may also cause delay in giving correct treatment, and may lessen the chances of success. This is particularly important nowadays, when recent advances in arterial surgery (Rob *et al.* 1956) have given these patients the chance of getting relief from pain.

The following ten case histories show that certain characteristics of the pain were common to all the patients, and that in each case the diagnosis was indicated by the clinical features. All the patients in this series were men of over forty years of age; in all, the blood Wassermann reaction was negative and examination of the urine showed no abnormalities.

CASE REPORTS

Case 1—An Englishman aged fifty-seven years attended in July 1952. He had suffered for three years from pain in both buttocks produced by walking 100 yards and relieved in a few minutes by rest. All pulses in both lower limbs were present, but the popliteal and tibial pulses disappeared when he exercised to the point of pain. The blood pressure was 160/90. The radiographic finding of very extensive calcification of the aorta supported the diagnosis of intermittent claudication due to partial aortic occlusion. Aortography was not performed.

Case 2—A Scotsman aged forty-nine years was seen in September 1952. He had suffered for four years from pain in the left buttock coming on after walking seventy yards and relieved in a few minutes by rest. All pulses in the left lower limb were absent. The blood pressure was 150/90. A radiograph of the pelvis showed calcification in the site of the left common iliac artery. Retrograde arteriography (Fig. 1) supported the diagnosis of claudication due to left common iliac occlusion.

Case 3—An Englishman aged forty-seven years was seen in September 1952. He had a severe paralysis of the left lower limb from poliomyelitis in childhood, the gluteus maximus being the only muscle that had escaped. For six months he had suffered pain in the left buttock coming on after walking 150 yards and relieved in a few minutes by rest. The pulses were

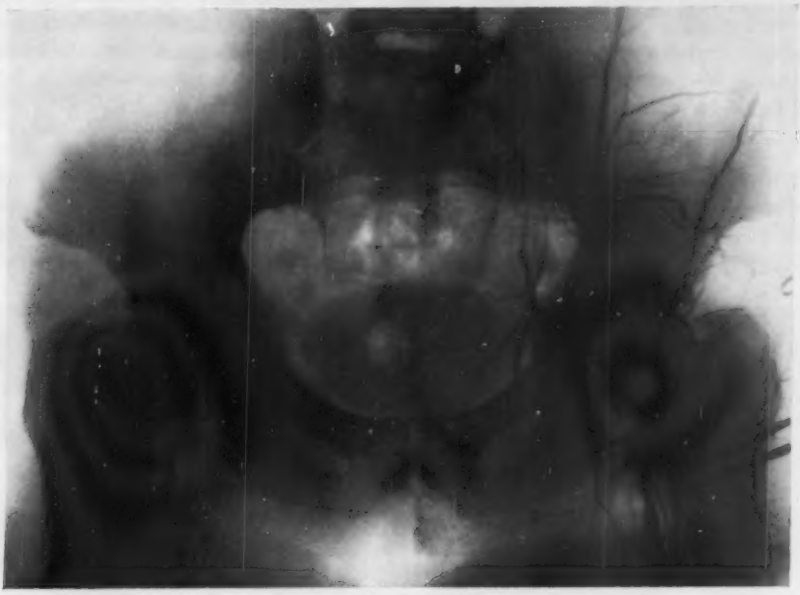

FIG. 1
Case 2—Retrograde iliac arteriography showing occlusion of the common iliac artery just proximal to its bifurcation.

FIG. 2
Case 4—Abdominal aortography showing diffuse arterial affection with partial occlusion of the left common iliac artery.

present, though diminished, in the affected limb, but became markedly reduced on exercise to the point of pain. The blood pressure was 145/100. Radiography showed calcification in the site of the left internal iliac artery. Aortography was about to be performed when he died at home from a coronary occlusion. No post-mortem examination was made, but the clinical and radiological evidence pointed to a diagnosis of partial common iliac or of internal iliac occlusion.

Case 4—An Englishman aged forty-seven years attended in January 1953. He had suffered for one year from pain in the left buttock radiating to the back of the thigh and leg, coming on after walking fifty yards and relieved in a few minutes by rest. All pulses were present in both lower limbs, but the left tibial pulses disappeared when he exercised to the point of

FIG. 3

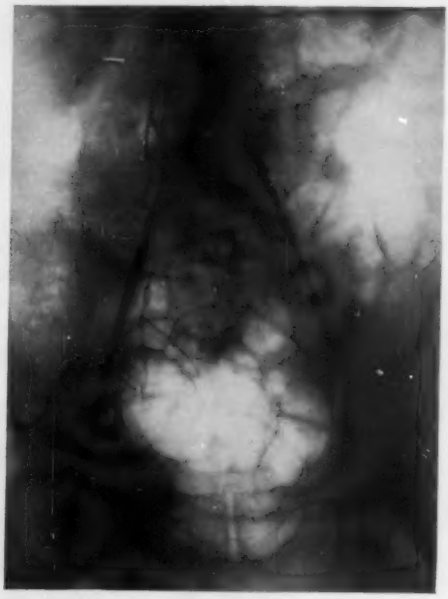

FIG. 4

Figure 3. Case 5—Abdominal aortography showing diffuse arterial affection with partial occlusion of the right common iliac artery and complete occlusion of the right external iliac artery. Note the large vertebral osteophytes. Figure 4. Case 6—Abdominal aortography showing nearly complete occlusion at the aortic bifurcation.

pain. Blood pressure was 210/100. Radiography did not demonstrate calcification in the site of the great vessels. Aortography (Fig. 2) showed diffuse arterial disease, with partial occlusion of the left common iliac artery.

Case 5—An Englishman aged sixty-eight years was seen in August 1954. He had suffered for several years from pain in the right buttock with radiation to the back of the thigh, coming on after walking 100 yards and relieved by rest. All pulses in the right lower limb were absent. The blood pressure was 180/110. Radiography showed a moderate amount of calcification in the site of the aorta and iliac vessels. Aortography (Fig. 3) showed partial right common iliac and complete right external iliac occlusion.

Case 6—An Englishman aged sixty-nine years attended in September 1953. He had suffered for five years from pain in both buttocks with radiation to the back of the thighs and to the calves, coming on after walking 100 yards and relieved in a few minutes by rest. Over the

past four years he had become aware of increasing impotence. All pulses were absent in both lower limbs, and the toes of both feet showed evidence of chronic ischaemia. The blood pressure was 130/85. Radiography showed no abnormal calcification. Aortography (Fig. 4) confirmed the diagnosis of occlusion at the aortic bifurcation.

Case 7—A Jew aged fifty-eight years attended in September 1952. He had suffered for one year from pain in the right knee and in the front of the right thigh coming on after walking 200 yards and relieved by a few minutes' rest. All pulses in both lower limbs were absent. The blood pressure was 160/80. Radiography showed moderate calcification in the site of the abdominal aorta. It is certain that this patient suffered from occlusion of the aortic bifurcation or of the iliac vessels, but he refused further investigation.

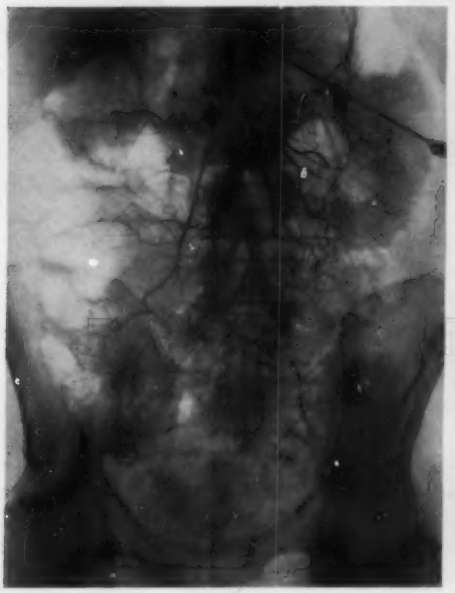

FIG. 5

FIG. 6

Figure 5. Case 8—Abdominal aortography showing complete right external iliac and partial left external iliac occlusion. The needle has entered the aorta rather high and this has resulted in filling of the superior mesenteric vessel. Figure 6. Case 9—Abdominal aortography showing complete left common iliac occlusion. In the later films good filling of the femoral artery was seen.

Case 8—A Jew aged fifty-six years was seen in June 1953. He had suffered for two years from pain in the front of both thighs, coming on after walking for about ten minutes, and relieved by rest. All pulses in both lower limbs were absent. The blood pressure was 150/80. Radiography showed no abnormal calcification. Aortography (Fig. 5) showed bilateral external iliac occlusion.

Case 9—An Englishman aged fifty-eight years was seen in September 1953. He had undergone a below-knee amputation in 1916 for a gunshot wound of the left foot. For two years he had suffered pain in the front of the left thigh coming on after walking 100 yards and relieved by rest. Pulses were absent in the affected limb. The blood pressure was 185/95. Radiography showed no abnormal calcification. Aortography (Fig. 6) showed a left common iliac occlusion.

Case 10—An Englishman aged sixty-four years attended in December 1955. He had suffered for one year from pain localised to the left buttock, which came on after walking for five

minutes and was relieved by rest. The left femoral pulse was only just perceptible, and the popliteal and tibial pulses on this side were similarly reduced. The blood pressure was 165/95. Radiography showed no abnormal calcification. Aortography (Dr David Sutton) showed a left common iliac obstruction.

DISCUSSION

It is the purpose of this paper to stress certain special points of importance in diagnosis, rather than to discuss the treatment of these cases.

1. All these patients were referred primarily to an orthopaedic department. All had previously had treatment on a diagnosis of an " orthopaedic " condition—osteoarthritis of the hip or lumbo-sacral disc degeneration.
2. Leriche (Leriche and Morel 1948) stated that the pain of aortic thrombosis differed from that found in true claudication, but the evidence from the present series and that of other workers (Holden 1946, Elkin and Cooper 1949, Ortner and Griswold 1950, De Bakey *et al.* 1954, de Wolfe *et al.* 1954) suggests that the pain differs only in site, not in character, from that affecting the calf in classical intermittent claudication.
3. The characteristics of the pain of vascular insufficiency were discribed and its mechanism was investigated by Lewis and his colleagues (Lewis, Pickering and Rothschild 1931). Their conclusion that the character of the pain is constant irrespective of its site, is well illustrated by the cases in the present series. A further good illustration of this point is furnished by the case of a man of sixty-eight years, seen in June 1952, who had pain of a " claudicant " nature in his right forearm resulting from an occlusion of the axillary artery. This constancy of the pain's character is indeed the most important single factor in making the diagnosis.
4. Gluteal and sciatic pain can be determined either by aortic or by common iliac obstruction. Pain in the front of the thigh can be determined by external iliac occlusion (Boyd and Jepson 1950), but it may also be caused by obstruction of the common iliac vessel.
5. Palpable tibial pulses do not exclude a diagnosis of vascular insufficiency in the lower limb. In three of the patients whose cases are described here, claudication was present with palpable tibial pulses. In all of these, the tibial pulses became impalpable after exercise to the point of pain. This " inverse response " to exercise of the pulsations in a limb affected by arterial disease was fully described by Ejrup (1948), and it is a test of great value in diagnosis.
6. Ischaemic changes in the peripheral parts of the limb are not necessarily a feature in these cases of proximal arterial obstruction. Only one of the ten patients studied here had marked peripheral ischaemic changes.
7. Radiography, by showing calcification in the site of the great vessels, is often a useful pointer to the diagnosis. It is suggested that in orthopaedic clinics, where attention is often directed largely to skeletal abnormalities seen on radiographs of the lumbar spine, closer search for evidence of aortic or iliac calcification might be rewarding.
8. The method of aortography used in these cases (Stripp 1954) does not require any expensive apparatus. It is a method of investigation which is simple enough to use and which, if care is taken, is not associated with any considerable hazards.

SUMMARY

1. Ten patients are described in whom pain due to arterial obstruction simulated pain caused by bone or joint disease or by disorder of the intervertebral disc.
2. The importance is stressed of arterial obstruction at the aortic bifurcation or in the iliac vessels as a possible cause of pain in patients attending orthopaedic clinics.

I would like to thank those members of the staff of the Royal National Orthopaedic Hospital who referred patients for examination, and especially Mr H. J. Seddon for the use of his beds for these investigations. I am grateful to Professor C. G. Rob and Dr David Sutton for their help, and to Mr G. C. Lloyd-Roberts for referring a patient (Case 4).

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LIPOMATOSIS OF SKELETAL MUSCLE IN MAFFUCCI'S SYNDROME

(Dyschondroplasia with Haemangiomata)

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The association of dyschondroplasia with angiomata was first described by Maffucci (1881). In 1942 Carleton, Elkington, Greenfield and Robb-Smith compiled a comprehensive review of eighteen reported cases and gave a detailed description of two additional cases. They suggested that the syndrome be associated with the name of Maffucci. Further examples have since been recorded by Krause (1944), Umansky (1946) and Strang and Rannie (1950), making a total of twenty-three published cases up to the present time.

Gross deformity associated with multiple chondromatous and angiomatous tumours has been a feature of the previously reported cases, but in our patient there has so far appeared only a single chondroma, and angiomata of skeletal muscle, subcutaneous tissue and periarticular tissue. A distinctive feature in our case was a tumour-like adipose infiltration of the semitendinosus muscle and short head of biceps femoris.

CASE REPORT

The patient, a boy, was born in May 1944 and was normal in all respects until the age of four years. His parents were not consanguineous, and there was no significant clinical history related to them or their one other child. A swelling was noticed by the mother on the medial aspect of the patient's right knee at the age of four. It was not painful, but was a little tender on pressure. The mass was excised, and histological examination proved it to be a haemangioma. The wound healed uneventfully.

However, a tense, firm tumour, about five centimetres in diameter, recurred at the same site two years later. At operation a cartilaginous mass was seen to arise from the tibial epiphysis. It was necessary to open the joint cavity in order to remove the tumour completely. The tumour histologically was a chondroma with angiomata in the periarticular tissues. The wound healed uneventfully.

Within four weeks the mother believed that there was a further swelling in the muscles of the back of the right thigh. Clinical examination revealed a slight flexion deformity of the knee but no tumour. The patient fell on the right knee eighteen months later, and the joint was then found to be irritable, and flexion severely limited. There was no radiological evidence of fracture. After the joint had been rested in a Thomas's splint for four weeks the acute condition subsided, but there was a fixed flexion deformity of 45 degrees. In February 1955, when the child was nearly eleven, a hard mass in the posterior thigh muscles was clearly palpable. At operation the tumour appeared to occupy the whole length of the semitendinosus muscle and the short head of the biceps femoris. There was some fibrosis of the surrounding connective tissue but no involvement of the sciatic nerve or other surrounding structures. The semitendinosus and short head of biceps were excised and the wound healed uneventfully. After the operation the range of movement of the joint improved, and after four months is now from 45 to 10 degrees. No further haemangiomata, chondromata or other abnormalities have been revealed by careful clinical and radiological examination.

Pathological features—*The first tumour removed from the right knee*—Two small blocks were available for histological examination. In both there were numerous rounded angiomatous nodules ranging from one to four millimetres in diameter (Fig. 1). Each nodule consisted

of a circumscribed mass of myxomatous fibrous tissue containing angiomatous vessels. The vessels resembled thick-walled capillaries with a prominent endothelial lining and sometimes without a lumen, but many were wider sinusoidal spaces filled with blood. Muscular arteries either within the nodules or in the closely adjacent tissues, showed marked fibro-elastic thickening of the intima. Macrophages contained a little haemosiderin.

The chondroma from the right knee—The block of tissue available for histological examination included a crescentic portion of the tumour, part of the joint cavity and the surrounding connective tissue. The tumour was a simple chondroma consisting of hyaline cartilage, with a well defined margin bounded by a fibrous capsule or perichondrium. That part of the tumour which extended into the joint cavity was covered by a thin layer of synovial tissue as well as by a thin layer of fibrous tissue continuous with the thicker perichondral capsule.

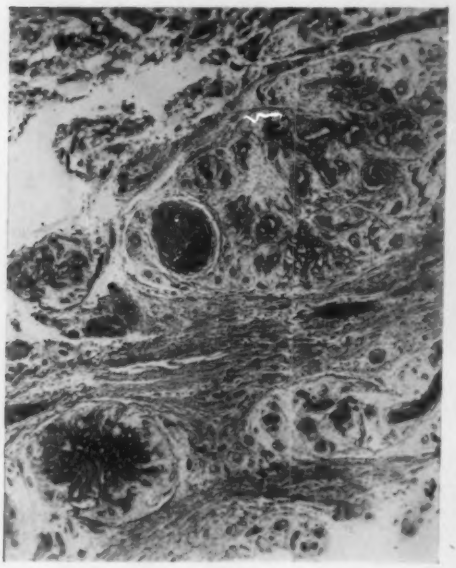

FIG. 1

FIG. 2

Figure 1—Angiomatous capillary vessels in pale rounded nodules of myxomatous fibrous tissue. Towards the centre is an artery with narrowing of the lumen from endarteritis obliterans. (Haematoxylin and Van Gieson, ×18.) Figure 2—Capillary and cavernous angiomatous tissue in the periarticular tissues. A thickened artery lies to the right. Part of the chondroma and synovial membrane are seen below. (Haematoxylin and eosin, ×17.)

The part of the synovial membrane reflected from the surface of the tumour appeared moderately oedematous and vascular. The attached periarticular tissues contained several capillary angiomata like those in the first tumour, and also two cavernous haemangiomata with thin-walled sinusoidal spaces surrounded by a moderate amount of fibrous connective tissue containing a little plain muscle (Fig. 2). A moderate amount of haemosiderin was found in macrophages in the surrounding connective tissue. Several related muscular arteries showed marked fibro-elastic thickening of the intima.

Semitendinosus muscle and short head of biceps femoris—The semitendinosus muscle was represented by a firm fusiform mass, twenty-five centimetres long and seven centimetres in diameter, invested by a thin fibrous fascia. It consisted mainly of compact, firm adipose tissue in elliptical compartments separated by thin fibrous septa. A broad tendinous structure was included near one end, but there was otherwise little resemblance to skeletal muscle

(Fig. 3). It contained many blood vessels with a varicose appearance, some of which were cut in longitudinal section for a distance of about three centimetres. Examination with a hand lens revealed that they consisted of compact aggregations of many small blood-filled spaces. The short head of biceps femoris measured eight centimetres in length and three centimetres in diameter, and had a similar appearance, but it more closely resembled skeletal muscle and appeared to contain less fat. Radiological examination of the specimens showed no opacities suggestive of phleboliths. Histological examination showed that the muscle compartments were almost entirely replaced by adipose tissue. The fibrous-tissue septa of

FIG. 3

The semitendinosus muscle consists chiefly of adipose tissue and contains many small angiomata. Some are cut in longitudinal section towards the top of the specimen, and at the opposite end there is a broad tendinous structure.

perimysium were increased in thickness and moderately oedematous. There were many discrete angiomata throughout the tissue, most of them about 0·25 centimetre across (Fig. 4). Each comprised a group of cavernous blood spaces, usually situated close to a muscular artery of medium size, and enveloped in a varying amount of plain muscle and fibrous connective tissue. The spaces were filled with blood and had generally a prominent endothelial lining. In many instances there were traces of an elastic lamina surrounding the angiomata, and occasionally it was intact round the whole circumference, an appearance suggesting phlebectasia (Fig. 5). Sometimes the walls of the angiomata contained vasoformative tissue showing budding endothelial tubes with or without a lumen. Thrombi were not seen in the angiomata, but occasionally haemosiderin was found in the surrounding connective tissue.

Relatively normal veins were seldom seen in relation to these lesions, whereas muscular arteries, often showing obliterative endarteritis, were almost constantly present.

The voluntary muscle bundles were everywhere severely atrophied. Surviving fibres were few and widely separated one from another by adipose tissue. They were more numerous close to the thickened perimysial bands (Fig. 6). Most of the striped muscle fibres were between 8μ and 15μ in diameter, but individual fibres near the perimysium measured up to about 80μ in diameter. Cross striation was preserved in most of the atrophic fibres (Fig. 7) but not infrequently pale, rounded, hyaline, and degenerated fibres were encountered with numerous sarcolemmal nuclei around them or within the substance of the fibre (Fig. 8). There was a little irregular lymphocytic infiltration and fibrosis of the endomysium, and some

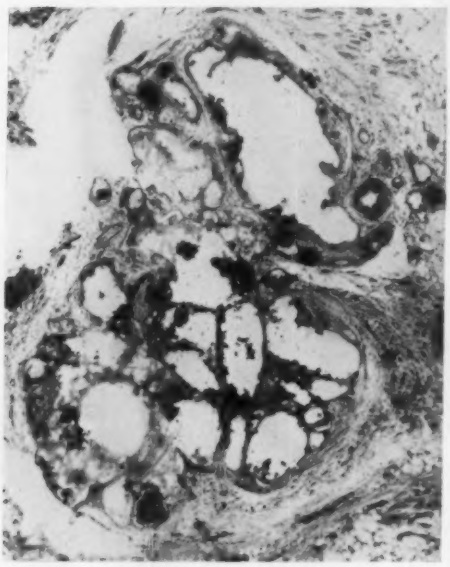

FIG. 4

FIG. 5

Figure 4—A cavernous angioma in the wall of which there is a small artery showing endarteritis obliterans. (Haematoxylin and eosin, ×36.) Figure 5—A phlebectactic vein resembling an angioma. It is surrounded by an intact elastic lamina and the lumen is intersected by fibro-elastic tissue. Adjacent muscular arteries show endarteritis obliterans of varying degree. (Weigert's elastin stain and Safranin, ×18.)

fibrous thickening of the walls of the smaller endomysial blood vessels. Follicular aggregates of lymphocytes were frequent, often several being encountered in a cross-section of a single atrophic muscle bundle.

DISCUSSION

Multiple chondromata and angiomata, with a tendency towards unilateral distribution, are the characteristic features of Maffucci's (1881) syndrome. The tumours usually appear during post-natal development and often cease to progress after puberty. The tumours that have so far appeared in our patient were limited to the right lower limb, and in our opinion this is a relatively mild case of the syndrome. Carleton *et al.* (1942) suggested that mild cases such as this might be overlooked and that further examples might be recognised if the manifestations of the syndrome were searched for in every patient with chondromata or angiomata. The findings of Umansky (1946), however, are not encouraging in this respect. He was able to find no instances of the syndrome, and no association of the two lesions, in sixty-five cases of dyschondroplasia and seventy-one cases of haemangiomata.

An important feature in our case was the angiomatous involvement of the semitendinosus muscle and short head of biceps. In most of the reported cases of Maffucci's syndrome the angiomata were soft purple tumours in the subcutaneous tissues, but in some they were deep-seated and extended down to bone. None appeared to originate primarily in muscle as in this case.

Skeletal muscle is a not uncommon site for angiomata. Jones (1953) found records of 358 cases in the literature and described fourteen further cases. They form only a small proportion of haemangiomata in general (Watson and McCarthy 1940), but according to Weaver (1938) most deep-seated angiomata of the lower extremities do arise in muscle. A total of 212 cases of angiomata in muscle, including eleven new cases of their own, were analysed by Davis and Kitlowski (1930), and they emphasised that the tumours were usually

FIG. 6

A section from semitendinosus muscle showing part of a cavernous angioma. A muscle bundle, lying between two thickened perimysial septa, is almost completely replaced by adipose tissue and most of the surviving muscle fibres lie close to the perimysium. In the centre of the field there is a follicular aggregation of lymphocytes. (Mallory's phosphotungstic acid haematoxylin, ×50.)

infiltrative, poorly circumscribed, cavernous haemangiomata, the muscle often seeming to contain multiple varicose veins, just as in the present case. Adipose tissue replacement of the muscle in the vicinity of the angiomata was frequently noted, and in two cases (Cases 2 and 7) of Davis and Kitlowski, approached in extent that observed in the present instance.

The adipose enlargement of the involved muscles in the present case was so pronounced as to give a misleading clinical impression of actual tumour. The angiomata in the muscle were in themselves little responsible for the swelling. The adiposity of the muscle appeared, as in pseudohypertrophic muscular dystrophy, to be secondary to marked atrophy of the muscle fibres. There was also considerable shortening of the muscles and flexion deformity of the knee, perhaps attributable to the perimysial and endomysial fibrosis observed histologically. Although atrophy and fibrosis of muscle have been found to follow disuse in dogs (Tower 1937, Reid 1941), only simple atrophy of muscle fibres is observed under similar conditions in

man (Adams, Denny-Brown and Pearson 1953), and the marked muscular atrophy and interstitial proliferation observed in the present case are not likely to be simply the result of splintage and disuse. Apart from the angiomata the pathological changes in the muscle are similar to those observed in the later stages of pseudohypertrophic muscular dystrophy. But there was no clinical evidence of a coexistent primary muscular dystrophy in this case, nor was there a family history of such a disorder. Especially in view of the similar fatty replacement in the two cases of angiomata in skeletal muscle described by Davis and Kitlowski (1930), we believe that the muscular atrophy and secondary adiposity and fibrosis must be directly connected with the angiomatous lesions present throughout the muscle mass. In our opinion these angiomata constituted multiple arterio-venous shunts and the resulting tissue anoxia was responsible for the pathological changes observed in the muscle. In fatty degeneration

FIG. 7

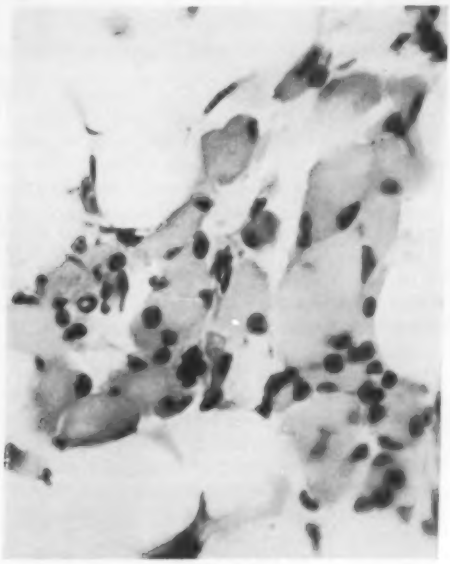

FIG. 8

Figure 7—Semitendinosus muscle. Atrophic muscle fibres, approximately 10μ in diameter, with intact cross striation. The muscle fibres are separated by adipose tissue. (Mallory's phosphotungstic acid haematoxylin, ×580.) Figure 8—Semitendinosus muscle. Pale swollen, hyaline and degenerate fibres with numerous sarcolemmal nuclei. (Haematoxylin and eosin, ×440.)

of heart muscle in anaemic anoxia the muscle fibres farthest away from the blood supply are the most severely affected. Similarly, in the case under discussion, the central fibres of the muscle bundles showed the most severe atrophy, whereas the peripheral muscle fibres, nearer the perimysium and its blood vessels, were relatively well preserved.

The local anoxia arising in this way would be persistent and of low grade, whereas most of the recorded experimental observations relevant to muscle ischaemia involve acute, and usually massive, vascular obstruction. Brooks (1922) produced severe fibrosis of skeletal muscle by venous obstruction and Griffiths (1940), by arterial obstruction, produced focal necrosis of muscle surrounded by a zone of fibrogranulation tissue. Both these authors considered those different pathological changes to be the essential pathological feature of the cases of Volkmann's ischaemic contracture they described. The pathological changes in the muscle observed in the present case do not resemble those described by either Brooks or Griffiths, and we consider that they are the result of persistent low-grade anoxia.

SUMMARY

A boy aged eleven with a solitary chondroma of the right tibia, and angiomata of the skeletal muscle, subcutaneous tissues, and periarticular tissues of the same limb, is considered to be a case of Maffucci's syndrome (dyschondroplasia with angiomata), although there was not the severe deformity encountered in the previously reported cases. There was a secondary atrophy and adiposity of skeletal muscle, and this was attributed to anoxic effects produced by the angiomata.

We wish to thank Mr F. W. Holdsworth and Professor D. H. Collins for much helpful advice in preparing this paper, and also Dr A. J. N. Warrack of the Sheffield City General Hospital for allowing us to use biopsy material from his laboratory.

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FRACTURES OF THE LATERAL TIBIAL CONDYLE TREATED BY SKELETAL TRACTION AND EARLY MOBILISATION

A Review of Sixty Cases with Special Reference to the Long-term Results

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When the straight knee is subjected to an abduction or valgus strain the medial ligament may tear or the lateral tibial condyle may fracture. This latter injury was first called a " bumper " or " fender " fracture by Cotton and Berg (1929) who considered the injury to be caused by the tyre or fender of an automobile striking the extended leg of the " jay-walking citizen." This mechanism has been accepted by some later authors (Cubbins *et al.* 1934, Fyshe 1952); but only five of the sixty patients in the present series were injured by a car. More recent work (Wilson and Jacobs 1952, Gylling and Lindholm 1953) has suggested that with the abduction force is combined compression produced by a fall, and this is indeed the more common mechanism. It would seem more logical therefore to call the injury a valgus split or crush, but one is loth to discard an accepted and picturesque term.

A bumper fracture may be defined as a fracture of the lateral tibial condyle caused by a combination of abduction and compression forces. The line of fracture runs downwards from the articular surface producing a simple split, or downwards and laterally separating a large fragment of the lateral condyle; this fragment may be depressed or tilted and occasionally comminuted. The fracture line may continue through the neck of the fibula, and the upper end of the fibula is then displaced with the tibial condyle. The abduction force damages not only the bone but also the medial ligament of the knee.

Bumper fractures have been subdivided in various ways. Gylling and Lindholm (1953) measured the downward shift of the lateral condyle, and Slee (1955), modifying Palmer's (1951) classification, divided the fractures into split fractures with little or no displacement, compression fractures with condylar depression, and T-shaped or Y-shaped fractures. It is difficult to subdivide bumper fractures precisely. I have tried, by estimating the displacement of the lateral condyle, to classify them into three groups: 1) mild, with little or no displacement; 2) moderate, with obvious depression or tilt of the lateral condyle; and 3) severe, with gross displacement or comminution.

TREATMENT BY IMMOBILISATION IN PLASTER

Most authors advocate closed reduction and immobilisation in plaster. The technique of reduction is usually a combined manoeuvre: traction to the leg, adduction at the knee and sometimes lateral compression. With more severely displaced fractures the force of such manipulations may be augmented by using a traction table and compression clamp (Watson-Jones 1955). A few authors advocate operative reduction if displacement is severe (Wilson and Jacobs 1952), sometimes with the addition of internal fixation by a screw (Perey 1952) or by wire or bone grafts (Jakobsen 1953).

Though debate and controversy continue on the disadvantages of open as against closed reduction, there seems to be unanimity on the method of maintaining reduction: namely, by fixation in plaster. The underlying assumptions are, presumably, 1) that osteoarthritis will inevitably follow a fracture into a joint, unless reduction is perfect and is perfectly maintained by rigid immobilisation until union is complete, and 2) that rigid immobilisation is necessary to permit the healing of associated ligamentous damage.

TREATMENT WITHOUT IMMOBILISATION

Most of the patients treated for bumper fractures at St Thomas's Hospital and at the Rowley Bristow Orthopaedic Hospital during recent years have had neither internal fixation nor plaster; instead they were treated by skeletal traction and exercises. Treatment without fixation was described by Perkins in 1940. Furlong (1953) and Fairbank (1954) gave eloquent testimony to the good results obtained without fixation, but, so far as I can discover, no detailed analysis of results has hitherto been published.

The main objects of this paper are to present the results of treatment by traction and exercises, to show that these are satisfactory, and to show that osteoarthritis does not, in fact, follow.

FIG. 1
Case 1—Bumper fracture in a man of fifty-two.

TECHNIQUE OF TREATMENT

The patient is anaesthetised and, if a haemarthrosis is present, the blood is aspirated. A Steinmann's pin is inserted one or two inches below the fracture. With severe displacement an attempt at reduction is made by applying traction through the pin and compression at the knee. The patient is returned to bed with his knee and calf resting on a pillow and with ten pounds' weight traction. The foot of the bed is raised on blocks.

Knee straightening and leg raising exercises are begun on the next day. Within a few days the patient should be able to raise his leg from the bed (Figs. 1 and 2). As soon as this has been achieved knee bending exercises are begun. In the average case the patient should have fully controlled extension, and flexion to at least 90 degrees, by the end of four weeks (Fig. 3).

At the end of six weeks the pin is removed and the patient gets up with crutches. Though he does not take weight on the affected leg, he goes through the normal movements of walking, so that the pattern of neuromuscular control may be more quickly regained.

Crutches are used for a further six weeks, but during the second three weeks of this stage he is able to take increasing weight on the leg. At the end of twelve weeks from injury he

walks unsupported and, unless he has the timidity and fragility of age, without a stick. The patient may return to sedentary work as soon as he gets up, and resume any other job as soon as he feels able.

No reference has been made to radiographs as a guide to treatment; they are probably irrelevant. Radiographs are taken to gratify the patient and for the academic value of confirming how often a good reduction has, by these simple methods, been achieved and maintained (Figs. 4 to 6).

CLINICAL MATERIAL AND ASSESSMENT OF RESULTS

The results in sixty patients with bumper fractures are presented. Their average age was fifty-three years. Twelve were treated by immobilisation in plaster (with the addition of screws and grafts in four cases). The remaining forty-eight patients were treated by traction and exercises as described.

The severity of injury was assessed radiologically, and, according to the degree of displacement, cases were classified as mild, moderate and severe. There has been no selection

FIG. 2

FIG. 3

Case 1. Figure 2—Seven days after the injury the patient is lifting his straight leg off the bed. Figure 3—Four weeks after the injury he can bend the knee to a right angle.

of cases, nor any significant difference in severity between the groups treated with and without fixation in plaster. Thus, of the twelve fractures treated by fixation in plaster, four were mild, five moderate and three severe. Of the forty-eight treated without fixation fifteen were mild, nineteen moderate and fourteen severe. When these figures are compared with those of other authors it seems likely that some cases that I have labelled " mild " would have been classified by others as " moderate." So far as can be ascertained, the cases presented differ from those in previously published reports only as regards the method of treatment.

In assessing the results the following terms were used.

Excellent—The knee was normal, did not interfere with the patient's work or play, did not ache or swell, and had a full painless range of movement.

Good—The knee was " barometric," aching in damp weather, or had slight limitation of full flexion, but was otherwise normal and did not interfere with work or play.

Fair—The knee hampered the patient slightly, ached after exercise, swelled sometimes and had a little limitation of movement.

Poor—The knee was worse than " fair."

The words used here in assessing results are widely used, but they are given different meanings by different authors. I have deliberately restricted " excellent " to those patients whose injured knee was as good as the other knee; if there was even slight restriction of

movement it was classified as " good." No patient whose knee was " excellent " or " good " has had to modify his work or hobbies because of the injury.

The success of any method of treating a fracture involving a joint must be assessed by the long-term results; so in the analysis that follows only the results of those patients examined more than five years after the fracture are considered in detail.

RESULTS OF TREATMENT WITH PLASTER

In the present series—Twelve patients were treated in plaster, only six being available for examination after five years. One result was excellent, two were good, two were fair and one was poor (arthrodesis was necessary).

This number is so small that analysis according to severity would be valueless.

Other published series—In considering the results reported by other authors one must allow for the fact that they may have been based on different criteria. Jakobsen (1953) described the results in sixty-five patients operated upon at the Uneval Hospital: thirty-five were excellent, sixteen good, four fair and ten poor. Perey (1952) described the results in 103 fractures: sixty-one were treated by plaster alone, twelve by operation and screw fixation, and thirty by operative elevation of the depressed condyle and bone pinning. Of these last thirty he was able to re-examine twenty-two after a sufficient interval. Eleven results were excellent or good, eight moderate and three poor. Gylling and Lindholm (1953) described the results in forty-seven cases in which operation was performed on those with severe displacement: six results were excellent, twenty-five good, thirteen satisfactory (this is taken to include patients who varied from " good " to " fair ") and three poor. They stated that secondary arthrosis occurred in 55 per cent and was more common in those operated upon.

RESULTS OF TREATMENT WITHOUT PLASTER

Forty-eight patients in the present series were treated by traction and exercises and without any form of fixation. Seven of these were not available for re-examination after one year and they are therefore omitted. In the remaining forty-one patients the results were: excellent twenty-two, good eleven, fair seven, poor one.

Long-term results—Twenty-seven patients were re-examined over five years after the injury (Table I).

TABLE I

DURATION OF FOLLOW-UP (TWENTY-SEVEN CASES)

Time	Number of cases
5–10 years	10
10–15 years	13
Over 15 years	4

The late results in these twenty-seven patients treated without fixation were: excellent sixteen, good five, fair five, poor one.

These twenty-seven long-term cases were further analysed according to the severity of the fracture (Table II).

Comparison with other reported series (Table III)—Accurate comparison between the series reported by different authors is not possible. Not only are the fractures subdivided in slightly varying ways according to type and severity, but, more important, the words used to express the results are given different meanings. Classification into " excellent," " good," " fair " and " poor " is a matter for judgment, and judgment is an art rather than an exact science.

DISCUSSION

The treatment of a bumper fracture by traction and exercises, without fixation, is simple and satisfactory. But the all-important question is: does osteoarthritis supervene? Gylling and Lindholm (1953) stated that degenerative changes occurred in 55 per cent of their cases, and there is no doubt that radiographs taken years after a bumper fracture sometimes show persistent depression of the lateral tibial condyle, with irregularity and diminished joint space on the outer side. These radiographic appearances do not, however, correspond in any way with loss of function or with pain. In other words, there may be no clinical evidence of osteoarthritis despite an abnormal radiographic appearance.

For example, a policeman aged thirty-six sustained a bumper fracture in 1942. In 1955 he was doing full duty at Buckingham Palace, with only slight "barometric ache," despite the

TABLE II

RESULTS FIVE YEARS OR MORE AFTER INJURY

Severity of fracture	Number of cases	Result			
		Excellent	Good	Fair	Poor
Mild	8	5	1	2	0
Moderate	11	7	2	2	0
Severe	8	4	2	1	1
Total	27	16	5	5	1

TABLE III

SUMMARY OF REPORTED RESULTS

	Author	Number of cases	Excellent	Good	Fair	Poor
With fixation	Jakobsen	65	35	16	4	10
	Perey	22	11		8	3
	Gylling and Lindholm .	47	6	25	13	3
	Present series . . .	6	1	2	2	1
Without fixation	Present series . . .	41	22	11	7	1
	Present series (after 5 years)	27	16	5	5	1

radiographs shown (Figs. 7 to 9). Again, a fireman of forty-six sustained a bumper fracture in 1945. To-day, eleven years later, he is still able to carry a 12-stone man up a ladder, and even to play football. Such examples are not unusual (Figs. 10 to 12). The joint appears to stand up well to hard wear over a period of years. In no case in the present series did the state of the knee alter, except for the better, after the end of one year.

It may be argued that holding the fracture immobilised in plaster might give still better results. The figures quoted do not support this contention. Out of twenty-seven patients treated by traction and exercises and followed up for more than five years, twenty-one gained either excellent or good results, using these terms in their strictest sense; only one result was poor. It may well be that, with a damaged lateral condyle, moulding by movement is the best way to achieve the greatest congruity of the joint surfaces.

FIG. 4

FIG. 5

FIG. 6

Case 2—Bumper fracture in a woman of sixty-two. Figure 4—Initial radiograph. Traction was applied for six weeks. Figure 5—Eight months later. Figure 6—Seven years later.

The method of traction and exercises permits movement without allowing abduction strain, so that any associated damage to the medial ligament is able to heal. Slight lateral instability does sometimes persist (though not as a disabling or troublesome symptom) whether the knee has been treated on traction or by plaster.

FIG. 7

FIG. 8

Case 3—Bumper fracture in a policeman aged thirty-six. Figure 7—Initial radiograph. Figure 8—Ten years later.

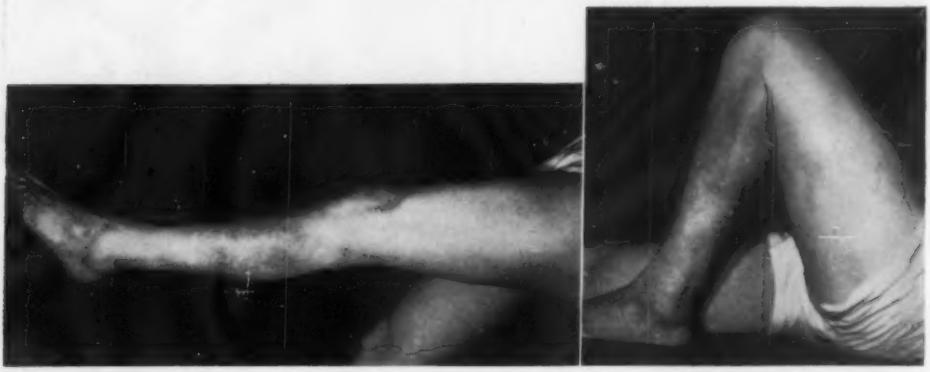

FIG. 9

Case 3—Despite the radiographic changes shown in Figure 8 the patient has a good range of knee movement and is on full duty.

It is doubtful whether plaster can be relied upon to hold a crush type of fracture successfully. In 1937 a woman of forty-eight sustained a bumper fracture; it was reduced, and held first in plaster and then in a caliper for a year. When she was re-examined in 1955 the original

FIG. 10

FIG. 11

Case 4—Bumper fracture in a man of seventy-seven. Figure 10—Initial radiograph. Figure 11—Five years later.

FIG. 12

Case 4—Appearance of knee and range of movement five years after the fracture.

valgus deformity was found to have recurred (Fig. 13). Prolonged splintage had failed to prevent the crushed bone from collapsing even after a year.

Fortunately any residual deformity after a bumper fracture is valgus, and a valgus knee from whatever cause hardly ever gives rise to clinical osteoarthritis. This is in striking contrast with varus deformity, which so often produces painful stiffness of the knee.

FIG. 13

Case 5—Bumper fracture after eighteen years. Despite reduction and splintage for a year the valgus deformity has recurred.

SUMMARY

1. The term " bumper fracture " is colourful but usually inaccurate. The injury is a valgus split or crush.

2. A series of sixty bumper fractures is reported: forty-eight were treated without operation or plaster.

3. Twenty-seven of the forty-eight patients treated without splintage have been followed up for more than five years, and seventeen of these for more than ten years.

4. The results are satisfactory and there is no evidence that there is any late deterioration of the joint.

5. It is suggested that bumper fractures should be treated without operation and without fixation in plaster.

I am glad to express my gratitude to Professor George Perkins, Mr R. J. Furlong and Mr F. A. Simmonds, who treated some of the cases presented, and without whose help and encouragement this paper could not have been produced.

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MALIGNANT TUMOUR AT SITE OF BONE PLATING

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The literature on the use of metals in bone surgery is now extensive and the effects of electrolysis and corrosion on the tissues have been studied by many writers. While much has been written about the damage to bone by the conjoint use of dissimilar metals, the surrounding soft-tissue reactions have received secondary attention. A search of the literature has failed to reveal any record of malignant changes from the use of metals in bone surgery or any suggestion that the possibility had ever been considered. This we believe is the first account of a malignant growth caused by tissue reaction at the site of a metal plate and screws used for the fixation of a simple fracture.

FIG. 1
Photograph of the arm showing swelling and marked discoloration.

CASE REPORT

A butcher, aged forty-two years, complained of a large discoloured swelling in the right upper arm (Fig. 1). The swelling had appeared about ten weeks previously and had increased in size gradually until a week before he reported, when there had been a sudden increase and the arm had become discoloured. There was no history of injury. At no time had he suffered any pain or discomfort and he was still at work. The swelling, which was soft and fluctuant in parts, extended from the origin of the deltoid muscle to the middle of the arm. The overlying skin was shiny and atrophic, and the swelling threatened to point anteriorly. The humerus, which could be palpated deep to the swelling, felt thickened and irregular. On the antero-lateral aspect of the arm was a scar three inches long; when questioned about it the patient gave the following story.

Thirty years before, when twelve years of age, he broke his right arm; the fragments were plated. Union occurred within a normal period and he was discharged from hospital six weeks after the injury. He had no further trouble with the arm until June 1949, when he fell on a slippery road with little force and was surprised to learn that he had broken his arm. The radiograph (Fig. 2) showed an oblique fracture through the middle of the humerus at the site of a screw hole. There was some bone absorption about the upper two screws and both were loose. A plaster was applied and retained for four weeks. After a spell in a broad sling he was able to resume work; clinical evidence of union was not checked radiographically. When questioned about the appearance of the arm after the fracture the patient stated that it appeared to him to be a bit thickened about the break and had remained so.

FIG. 2

FIG. 3

FIG. 4

Figure 2—Radiograph of the fracture sustained in 1949. There is considerable reaction round the loose upper screws. The plate was inserted in 1924. Figure 3—Condition of humerus when patient reported in 1954. Figure 4—Soft-tissue radiograph showing the site of the swelling in relation to the plate and screws.

A radiograph taken when we first saw him (Fig. 3) showed an ununited fracture of the middle of the humerus with sclerosis and overgrowth of bone. The plate was shown detached and angled away from the humerus at its upper end; the screws were loose and the top one lay in the soft tissues. Figure 4 shows the soft-tissue swelling over the upper arm.

No definite diagnosis of the swelling was made. Clinically it resembled a haemangiomatous type of lesion; but it was agreed that it was due to the presence of the plate and screws and that these should be removed and a biopsy performed. Several weeks passed before the patient was admitted, but the arm remained symptomless and looked the same.

Operation—The humerus was approached through a curved lateral incision, behind the swelling. After the biceps had been retracted medially the swelling, which did not involve bone, was stripped from the humerus, and the uppermost screw at the top of the plate was exposed; the

lower end was buried in osteoid tissue, and broke while being removed. The plate and screws were blackened and corroded (Fig. 5), both bone and soft tissues were discoloured, and a dark serous exudate was present around the area. The swelling was soft and friable and bled freely, and it was now obvious that it was a highly vascular malignant growth involving the soft

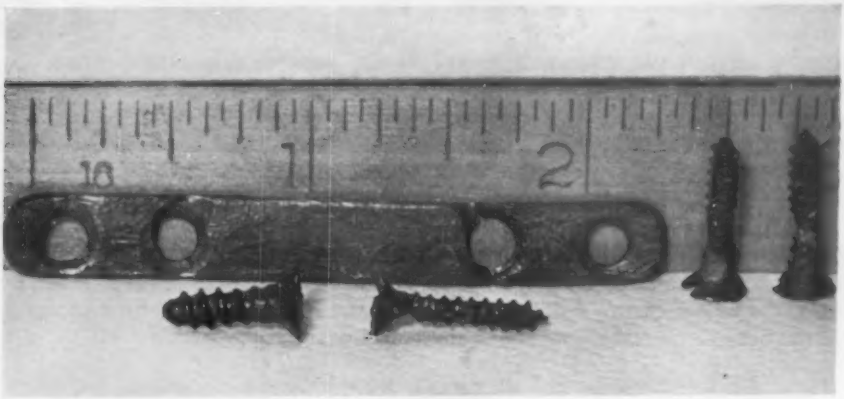

FIG. 5
Extensive corrosion of plate and screws. The screws are eroded at the point of contact with the plate.

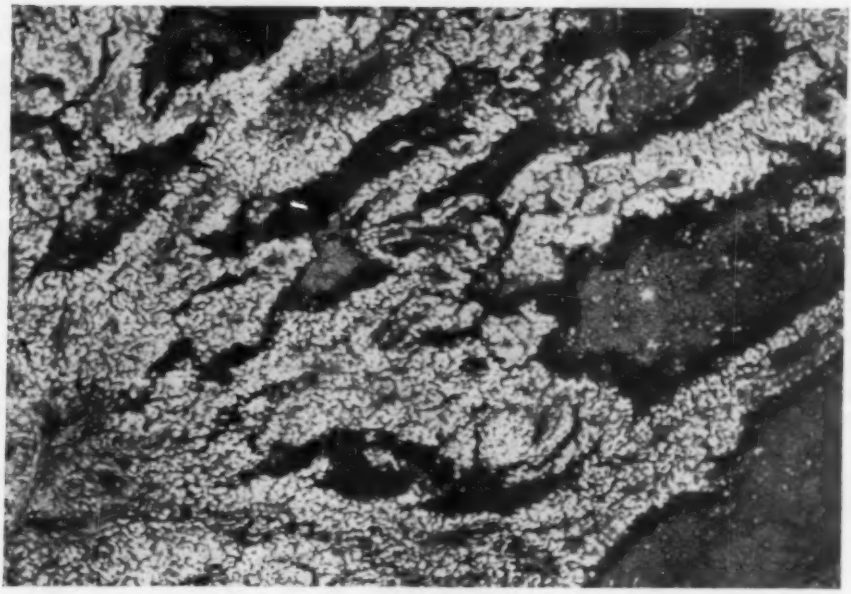

FIG. 6
Low-power photomicrograph showing closely packed cells lying in vascular spaces.

tissues. When a piece was removed for examination bleeding became profuse, and two pints of blood were given during the course of the operation. A piece of bone was also taken from the fracture site.

Pathology—Dr R. McAndrew reported that the soft-tissue specimen consisted entirely of a malignant neoplasm of uncertain type. It was largely necrotic and haemorrhagic. In the

less necrotic areas it sometimes resembled a carcinoma, but more often a tumour of mesoblastic origin, for instance an endothelioma. The cells were polygonal and closely packed and lay in vascular spaces. The most probable diagnosis was a sarcoma of the Ewing type (Fig. 6). In the bone section there was evidence of old injury but none of tumour.

Analysis of plate and screws—The plate and screws were analysed by Dr J. McLeod of the Royal Technical College, Glasgow, who found that the plate consisted of stainless steel and that its composition was: iron 74 per cent, chrome 18 per cent, nickel 8 per cent. The screws were composed of iron 88 per cent, chrome 12 per cent, carbon a trace. There was a difference in potential of 80 millivolts between the plate and screws.

Progress—The patient refused an interscapulo-thoracic amputation: the arm was painless, he could use it and he required both hands for his job. The tumour diminished considerably with a course of deep x-ray treatment. Radiographic examination of the chest and skeleton revealed no evidence of dissemination and the patient was allowed to return to work.

He was reviewed periodically and remained in good health and at work until a year after he was first seen, when he began to lose weight and feel tired. Two months later there was clinical evidence of lung metastasis, and a month later the liver was enlarged and tender; the abdomen contained free fluid. He died a few weeks later. No necropsy was possible. There had been no clinical change in the appearance of the arm.

DISCUSSION

When the pathological report was received a complete investigation was carried out to exclude the possibility that this might be a secondary deposit, but there was no clinical or radiological evidence of a growth elsewhere.

The possibility that the tumour had arisen independently was discussed. That a malignant growth should appear fortuitously at this site seemed most improbable, because there was radiological evidence of electrolytic changes five years before, and, from the patient's description of the arm, soft-tissue changes were probably present as well.

The plate and screws showed signs of corrosion and there was a difference in potential, sufficient to act as a source of irritation for close on thirty years. Malignant disease at the site of metallic foreign bodies in the lung was reported by Siddons and MacArthur (1952). In their two cases, thirty-two and thirty-five years respectively elapsed before the appearance of the malignant changes. Jones and Lieberman (1936) noted the marked soft-tissue changes that occurred around iron plates used experimentally in animals. Scales and Zarek (1955) issued a reminder that iron and chrome react with the protein molecule. Schinz and Uehlinger (1942) were able to produce sarcomatous changes in animals with chrome implants, and Hueper (1952) produced malignant changes with nickel. It is impossible to state in this case whether the tumour arose because of the presence of a single metal with carcinogenic properties, or because of the conjoint use of dissimilar metals.

The period of thirty years which passed between the insertion of the metals and the appearance of the malignant change accords with the observation by Willis (1948) that usually there is a long latent period between the application of the carcinogenic stimuli and the development of the tumour.

SUMMARY

1. A case is described in which a malignant tumour developed in the soft tissues at the site of a bone-plating operation performed thirty years before.
2. The plate and screws were found to be composed of dissimilar metals and a difference of potential existed between them.
3. A careful consideration of the history and clinical course indicates that the tumour arose because of the presence of the metals.

I wish to express my thanks to Mr James Patrick, who had charge of this case, to Dr J. Leith for the colour photograph and to Mr W. Towler, clinical photographer at the Glasgow Royal Infirmary, for the remaining reproductions.

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NON-OSTEOGENIC FIBROMA OF BONE

(Fibrous Metaphysial Defect)

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The term non-osteogenic fibroma of bone was introduced by Jaffe and Lichtenstein in 1942 to describe a distinctive benign lesion occurring near the ends of the long bones in young people. The excellent prognosis of this condition is sufficient reason for separating it from other more serious diseases of bone with which it has previously been confused. Reports in the American literature in recent years seem to indicate that this lesion is far from rare, yet

FIG. 1
Case 1—Initial radiographs of left tibia.

accounts of similar cases are hardly to be found in the British literature. Though there is general agreement over the radiological and histological features of non-osteogenic fibroma of bone, there is still uncertainty as to the nature of the disorder. For these reasons the following case reports and observations may be of interest.

CASE REPORTS

Case 1—A boy aged ten years complained of pain in the left leg which had started six weeks previously without antecedent injury or illness. He also noticed a swelling on the outer aspect of the leg. There

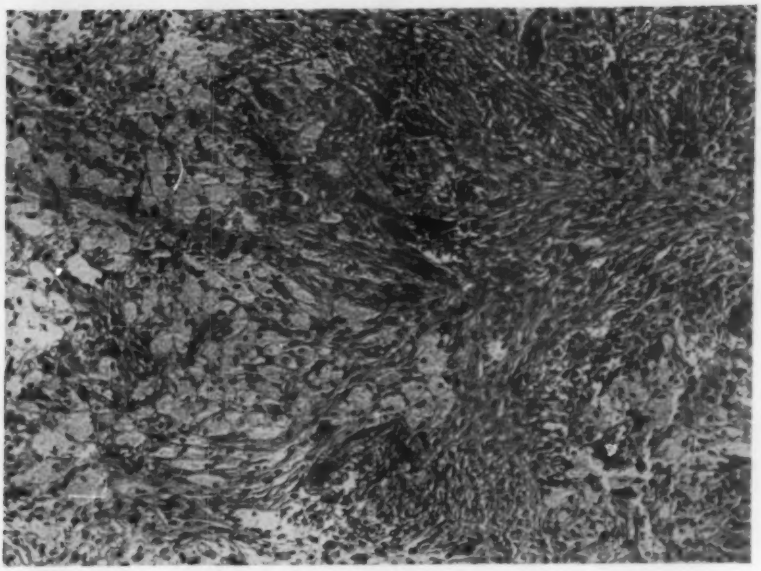

FIG. 2

Case 1—Biopsy tissue showing collections of foamy macrophages against a background of spindle-shaped fibroblasts, with small multinucleate giant cells. (×200.)

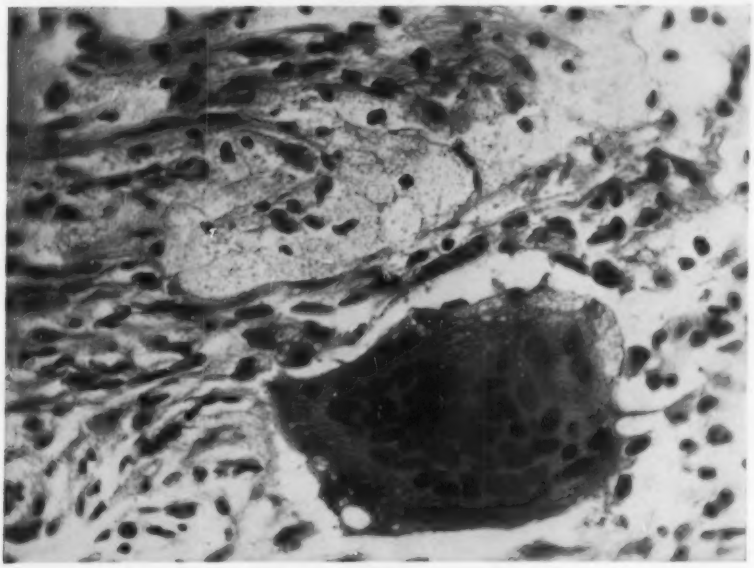

FIG. 3

Case 1—An osteoclastic giant cell of larger size than average, together with spindle cells and foam cells. (×480.)

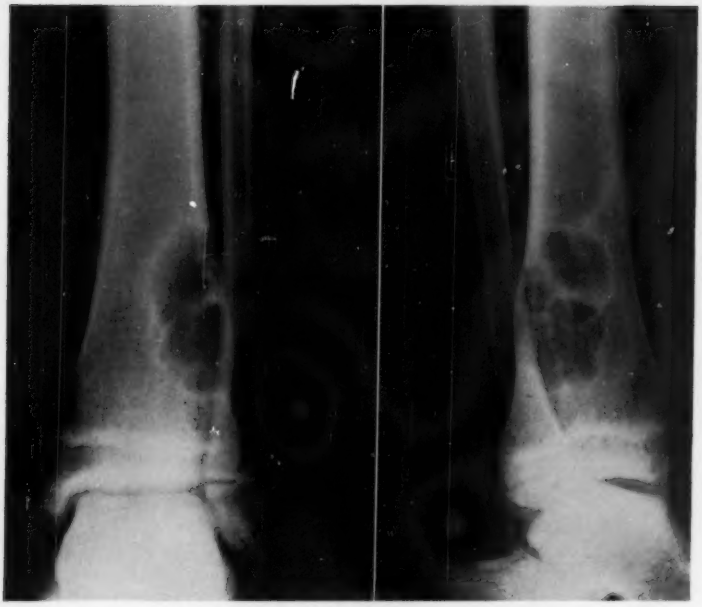

FIG. 4
Case 2—Initial radiograph of left tibia.

FIG. 5
Case 2—Skeletal survey revealed further lesions in the upper tibia and lower femur of the opposite limb.

was no other relevant medical history. On examination he appeared a normal, healthy boy for his age. There was a slight thickening on the antero-lateral aspect of the lower third of the left leg. It was two inches in diameter, apparently fixed to the bone, neither fluctuant nor tender, and the overlying skin was normal and not adherent.

Radiographs showed an irregular cavity in the outer half of the lowest third of the tibia. The general shape was oval, the longer axis being in the long axis of the tibia. The antero-medial border was sclerotic and the cortex on the posterior aspect was expanded. An appearance of loculation was present (Fig. 1). No other abnormality was found on skeletal survey.

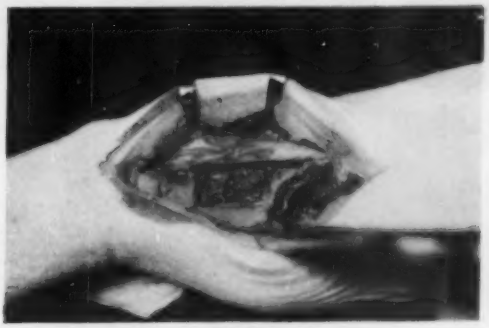

FIG 6.
Case 2—At operation a window was raised and reddish-brown, soft, friable material, mottled with yellow flecks, was present.

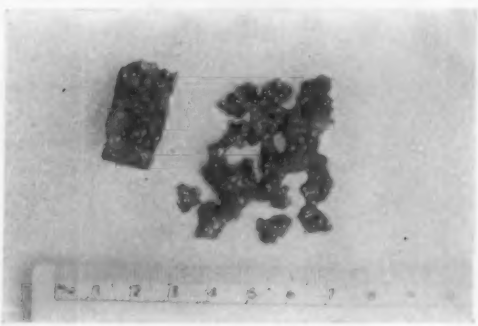

FIG. 7
Case 2—Material removed at biopsy.

Investigations—Mantoux reaction negative. Erythrocyte sedimentation rate 4 millimetres in 1 hour. Blood leucocyte count within normal limits.

Operation—Exploration revealed no definite soft-tissue swelling, but the outer aspect of the tibia was slightly expanded. The cavity in the bone, which measured $3 \times 2 \times 1$ centimetres, was found to contain soft, yellowish tissue which resembled inspissated pus. The cavity was widely opened and saucerised and, after removal of the contents, the lining was carbolised. The material was sterile on culture.

Pathology—On histological examination the specimen was found to consist of rather cellular connective tissue mingled with osteoclastic giant cells and foamy macrophages. There was no evidence of new bone formation. A diagnosis of non-osteogenic fibroma of bone was made (Figs. 2 and 3). The patient made a satisfactory convalescence and there has been no recurrence of the lesion.

Case 2—A girl aged twelve years complained of pain of insidious onset in the lower left leg. There was no history of injury. On examination there were slight swelling and tenderness over the lowest third of the tibia. Ankle movements were full.

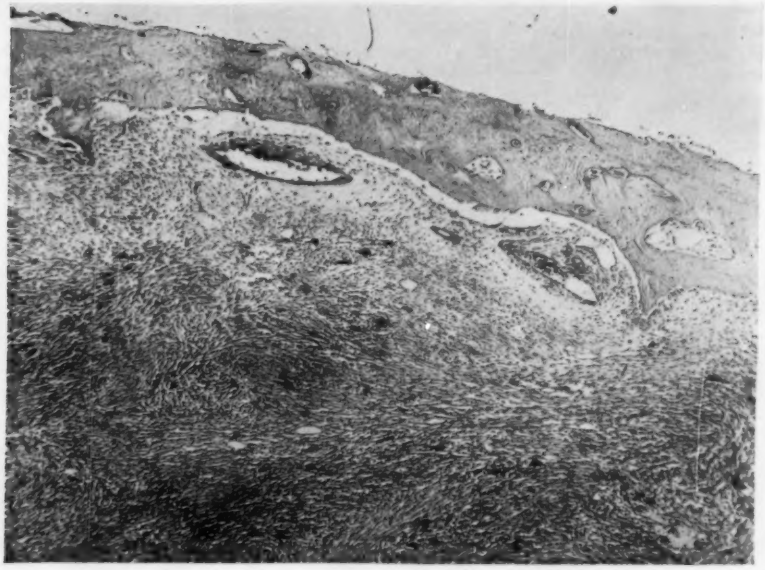

FIG. 8
Case 2—The thinned tibial cortex with the underlying lesion. Note the absence of new bone within this tissue. (×30.)

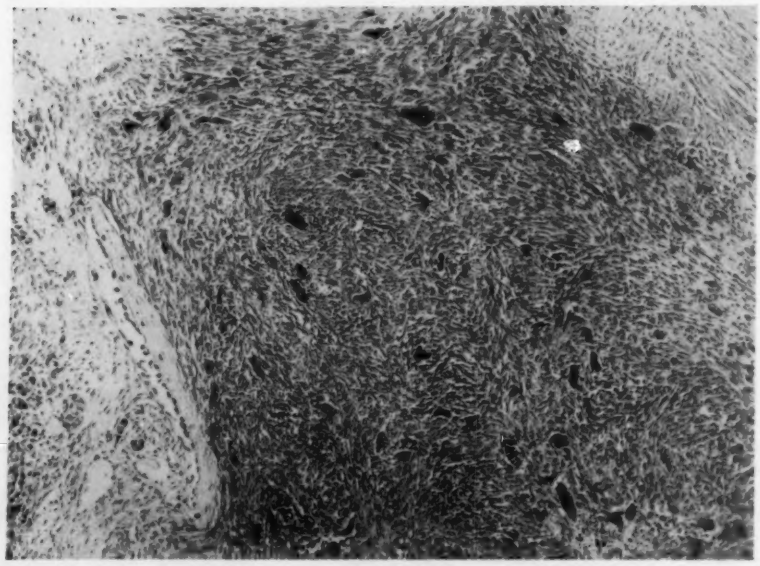

FIG. 9
Case 2—Edge of a loculus showing a slender bone trabecula in the septal wall. (×70.)

Radiographs showed a multilocular defect in the postero-lateral aspect of the lowest third of the tibia (Fig. 4).

A walking plaster was applied for two weeks. She improved for a time, but six months later the pain recurred. At this time further skeletal radiographs revealed somewhat similar, though much smaller, defects in the lower end of the right femur and the upper end of the right tibia (Fig. 5).

Operation—Postero-lateral approach. A bulge with a bluish tinge was present in the cortex posteriorly. A "window" was removed and the whole contents of the lesion were curetted out. The material was soft and friable, reddish-brown in colour with yellow flecks (Figs. 6 and 7). The cavity was packed with bone chips.

Histological examination showed a cellular connective tissue with plump, spindle-shaped fibroblasts and rather numerous multinucleate giant-cells. No foam cells were found in this case. The central parts of the several loculi were richly cellular and well supplied with thin-walled capillary blood vessels. In this tissue there were numerous extravasated red cells, and the fibroblasts, but not the giant cells, gave a strongly positive Prussian blue reaction for free iron. Beneath the eroded cortex and bordering the thin bony septa which formed the walls of the loculi, the tissue was less cellular and more fibrous in character. There was no evidence of new bone formation within the lesion itself and the few trabeculae traversing the area consisted in the main of lamellar bone (Figs. 8 and 9).

FIG. 10
Case 3—Fracture through multilocular "cyst."

Case 3—A boy aged seventeen experienced a sudden pain in his left arm while throwing a football. He had not felt any pain before in this arm and had no other symptoms. On examination there was an obvious deformity in the left arm.

Radiographs revealed an oblique fracture running through a multilocular, translucent area in the uppermost third of the humerus (Fig. 10). This lesion was oblong with the long axis in the long axis of

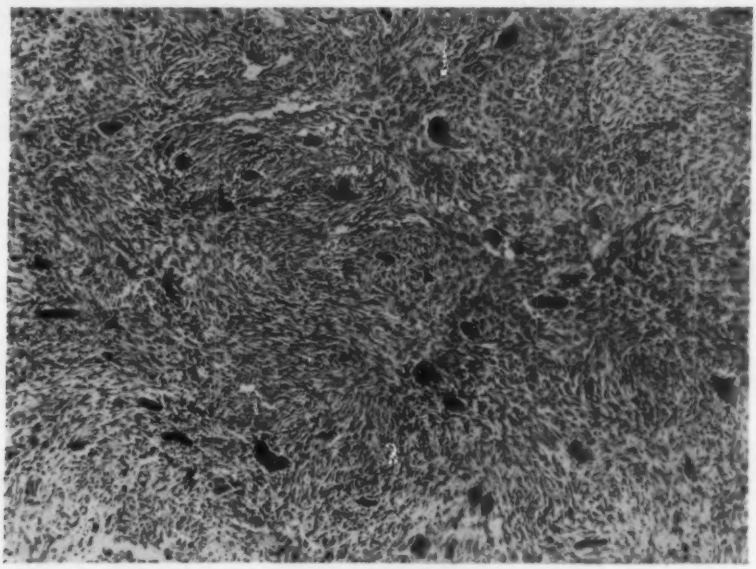

FIG. 11
Case 3—Cellular area showing plump, spindle-shaped fibroblasts with rather numerous giant cells of typical appearance. (×100.)

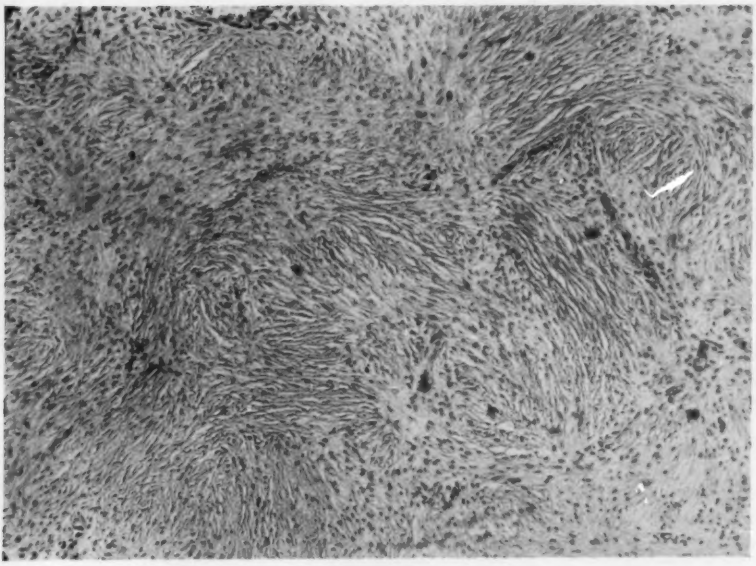

FIG. 12
Case 3—A less cellular area showing interweaving bundles of fibrous connective tissue. (×100.)

FIG. 13
Case 3—Dense accumulations of foam cells interspersed with strands of collagenous connective tissue. (×100.)

the bone. It was surrounded by a thin sclerotic margin. A diagnosis of fracture through a simple bone cyst was made on the radiographic evidence. The arm was at first immobilised, but operation was later performed.

Operation—Anterior approach. The cortex forming the anterior wall of the humerus at the level of the lesion was bulging slightly and when opened was found to be thin. The " cyst " contained solid yellowish tissue of a rubbery consistency. This was removed, the wall was carbolised and the cavity packed with iliac bone chips.

Histological examination showed fibroblastic tissue of varying degrees of cellularity, in which scattered multinucleate giant cells and collections of lipoid-filled phagocytes were present (Figs. 11 to 13). New bone formation was not observed within this tissue, though normal callus was found in relation to the fractured cortex.

The histological appearances were at this time interpreted as those of fibrous dysplasia of bone, but, on review, the diagnosis was amended to " non-osteogenic fibroma of bone."

Progress—The patient made an uneventful convalescence and radiographs taken eight months later revealed that the fracture had united and, in the cystic area, there was incorporation of the bone grafts.

FIG. 14

Case 4—" Healing " lesion in femur, with less definite loculation and more sclerosis than in younger patients.

Case 4—A youth aged nineteen complained of having had pain in the right knee for two months. There was no history of injury. On examination slight tenderness and swelling were found over the right tibial tuberosity, and there was slight tenderness over the lower end of the femur, but no swelling. Knee movements were full. A diagnosis of Osgood-Schlatter's disease was made.

Radiographs revealed a multilocular swelling in the lower end of the right femur on the outer side, encroaching upon, but not expanding, the cortex. The lesion itself was fairly dense and it had a sclerotic inner margin (Fig. 14). No further abnormalities were seen in a skeletal survey.

Operation—Postero-lateral approach. The cortex was thick and no distinct cavity was present. Within the dense bone were two or three small orange-yellow foci, each about two millimetres in diameter.

Histological examination of the excised window of the cortex showed several small loculi towards the inner aspect, each occupied by rather cellular connective tissue having a whorled pattern (Fig. 15). Multinucleate giant cells were absent, but there were conspicuous clusters of foamy macrophages. The loculi were enclosed by compact bone, mostly of lamellar type, though some woven bone was present on the inner aspect of the bony shell (Fig. 16).

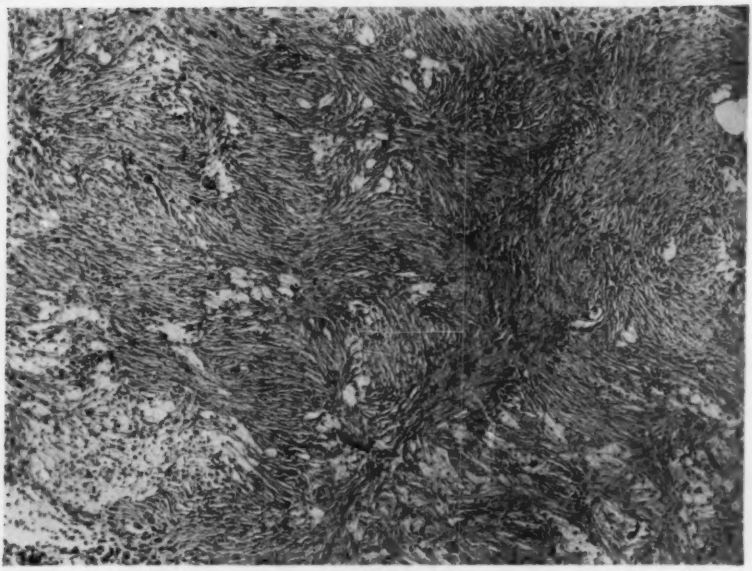

FIG. 15
Case 4—Small groups of foam cells in whorled fibrous connective tissue. (×70.)

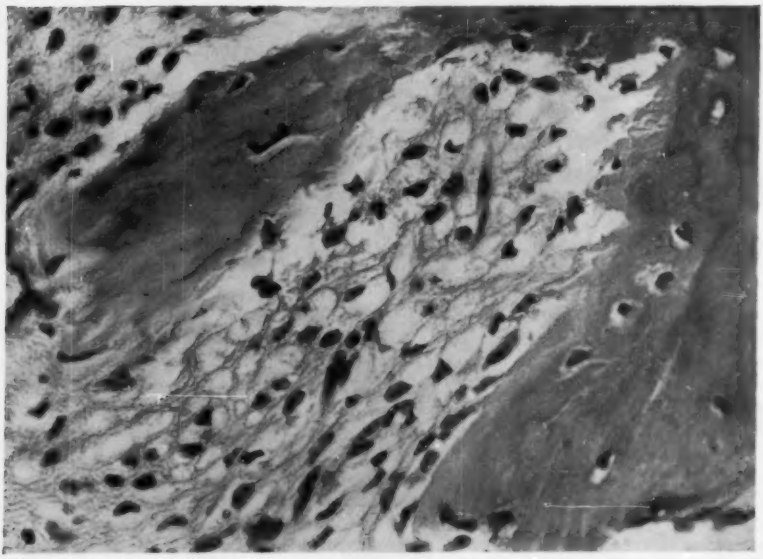

FIG. 16
Case 4—Woven bone forming on inner aspect of cortex. (×480.)

FIG. 17
Case 5—Initial radiograph. Fracture through " cyst."

FIG. 18
Case 5—After a year and a half.

FIG. 19
Case 5—After three years.

FIG. 20
Case 5—After four years.

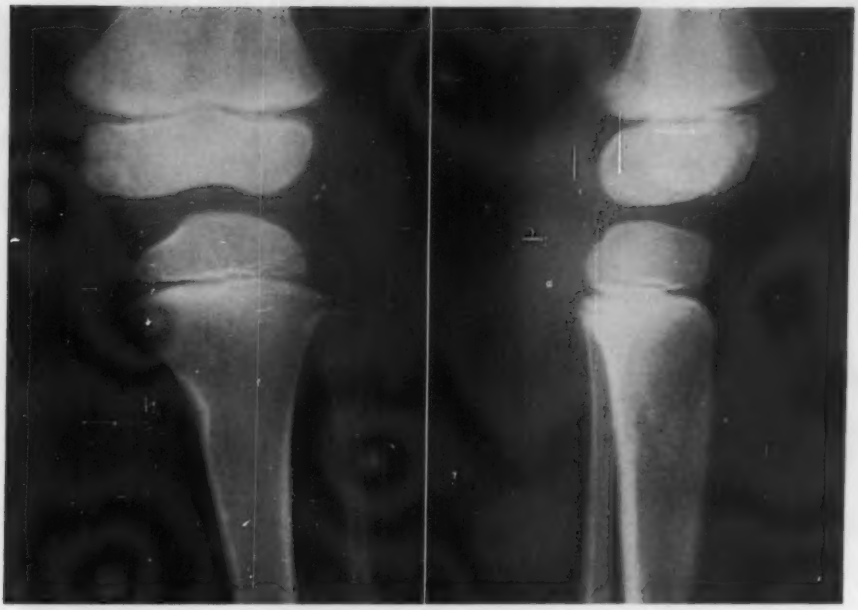

FIG. 21
Case 6—Defect in upper tibia of a boy aged four.

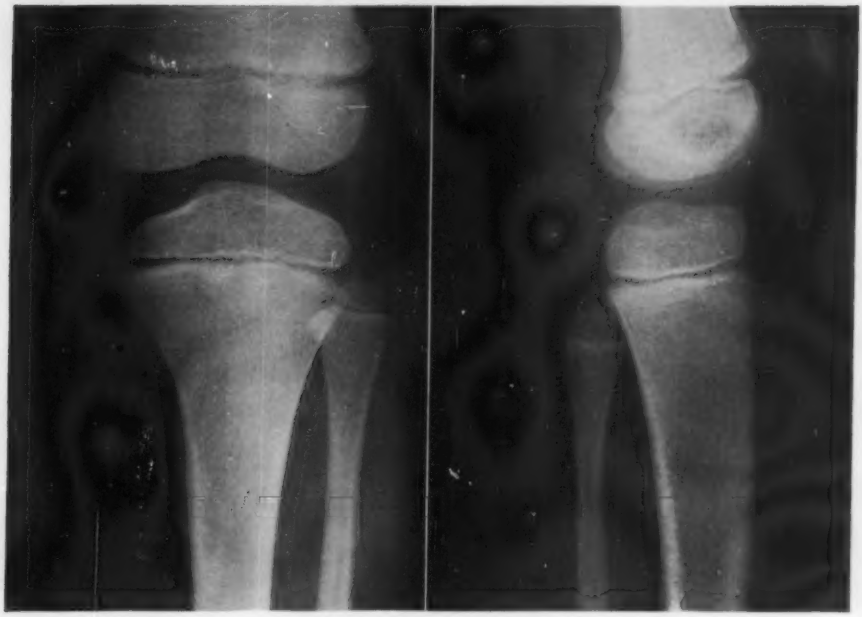

FIG. 22
Case 6—Two years later the defect is not present.

Case 5—A boy aged fourteen years complained of pain after twisting his left ankle; he had not felt any pain previously. On examination the lower third of the leg was swollen and tender.
Radiographs showed a fracture through a " cyst " in the lower end of the tibia on the lateral aspect (Fig. 17). The " cyst " was roughly oval, eccentrically placed in the bone three centimetres from the lower tibial epiphysis. The longer axis lay in the long axis of the tibia and was approximately four centimetres in length. It appeared to be multilocular and the overlying cortex was thin and " expanded." A fracture line ran obliquely through the " cyst " and there was minimal displacement.

He was treated in a walking plaster for eight weeks, and made an uneventful recovery. Biopsy was not performed. The lesion has not given rise to any further symptoms, but radiographs have been taken at intervals over the succeeding four years (Figs. 17 to 20). These show that the " cyst " has travelled away from the epiphysis with the growth of the bone and has moved outwards into and through the cortex. During the period of observation the lesion has not enlarged, but the edges have shown increasing sclerosis with accentuation of the septa between the loculi. In the final radiograph (Fig. 20) the outlines of the defect have become fainter.

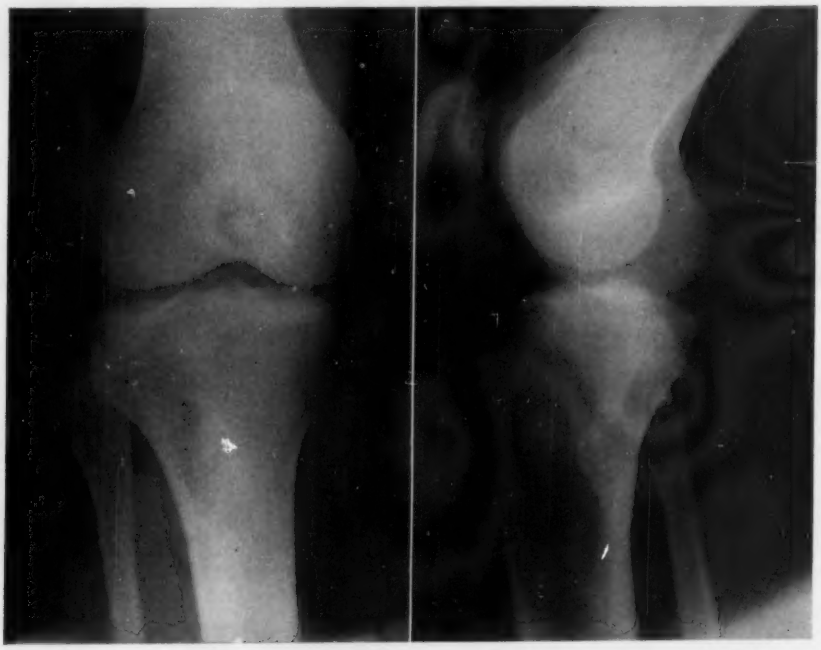

FIG. 23
Case 7—Lesion in the tibia of a man aged twenty.

Case 6—A boy aged four complained of slight aching in the region of the left knee. On examination no physical signs were present, but radiographs revealed a small defect in the upper end of the tibia on the medial side. The pain subsided spontaneously and radiographs taken two years later revealed no trace of the lesion (Figs. 21 and 22).

Similar bony defects were incidental findings in four further patients in whom radiographs were taken after minor injuries. In none of these were there symptoms relating to the lesion and biopsy was not performed.

Case 7—A man aged twenty complained of pain in the middle of the right leg radiating to the dorsum of the foot. Apart from slight tenderness of the anterior tibial muscles there were no physical signs. Radiographs revealed a multilocular lesion in the postero-lateral part of the upper third of the right tibia (Fig. 23). This was not thought to be responsible for his symptoms, which soon subsided.

Case 8—A girl aged twelve fell and sustained a crack fracture of the patella. Radiographs showed, in addition to the fracture, a translucent lesion in the lower femur close to the medial cortex (Fig. 24). Biopsy was not performed. She made a satisfactory recovery and now has no symptoms.

FIG. 24
Case 8—Lesion in the femur of a girl of twelve.

FIG. 25
Case 9—Lesion in the tibia of a boy aged eleven.

Case 9—A boy aged eleven and a half years fell and sustained a contusion of his left knee. Radiographs revealed a translucent, eccentric and circumscribed lesion in the upper third of the tibia (Fig. 25). The distal part was denser than the proximal and even more dense than the surrounding normal bone. He made a rapid and uneventful recovery and biopsy was not performed.

Case 10—A man aged twenty-two twisted his left ankle. Radiographs revealed a multinuclear lesion in the lower third of the left tibia (Fig. 26). There was no previous history of pain. He made an uneventful recovery from his injury and biopsy was not performed.

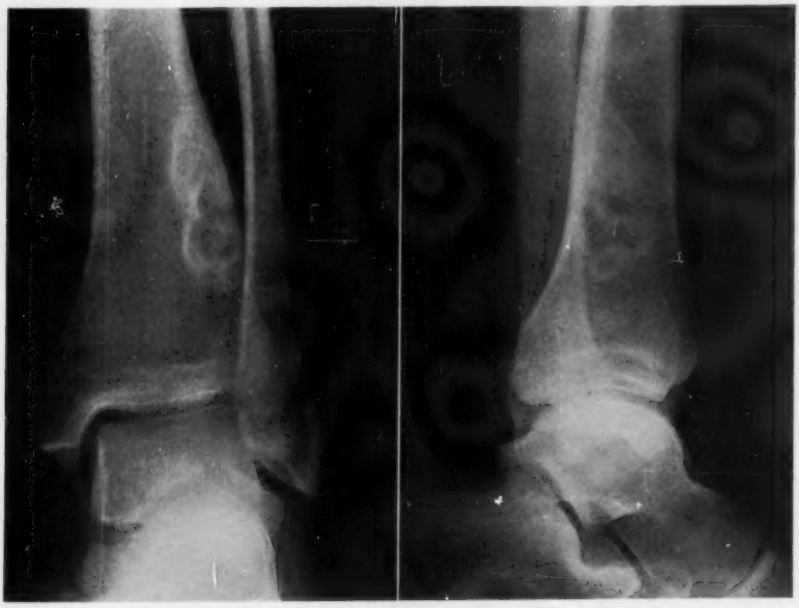

FIG. 26
Case 10—" Healing " lesion in the tibia of a man aged twenty-two.

DESCRIPTION

Non-osteogenic fibroma of bone is seen mainly in older children and adolescents and only rarely after the age of thirty. Both sexes are affected. The lesion occurs near the end of a long bone, most frequently in the lower limb. The commonly recorded sites are: lower end of femur, both ends of tibia and fibula, upper end of humerus, and lower ends of radius and ulna. Sometimes more than one bone is affected.

Though pain or a pathological fracture may lead to the discovery of this condition, it is frequently symptomless and only found after radiographic examination for some other reason. The radiographic appearances of non-osteogenic fibroma are distinctive. It appears as a rather sharply defined, translucent area, usually giving an impression of loculation and commonly bordered by a narrow zone of increased density. Its long axis lies in the long axis of the bone and at times exceeds four centimetres. Even when the lesion is large it tends to be eccentric in the bone, " hugging the cortex," as Jaffe and Lichtenstein (1942) expressed it. In some instances the translucent area is seen to lie within the expanded cortex, over which periosteal new bone may have developed. Only in slender bones such as the fibula or ulna does it commonly extend across the whole width of the shaft. When first discovered, the defect is usually separated from the epiphysial cartilage by two centimetres or more of apparently normal bone.

At operation a thin-walled cavity is found, sometimes multilocular, though the loculated

appearance in radiographs may be due to the persistence of bony spurs on the inner aspect of the eroded cortex. The cavity does not contain fluid but is filled with soft and semi-diffluent or firm and rubbery material, reddish-brown, yellow, or mottled in colour.

Histologically, the material is composed of connective tissue of varying cellularity. In the richly cellular areas small multinucleate giant cells are scattered among the spindle-shaped fibroblasts. There may also be groups of small round cells which Hatcher (1945) has described as lymphoblasts. This type of tissue is very vascular and in it may be found extravasated red cells and haemosiderin pigment which contribute to the reddish-brown colour of the lesion. Elsewhere the tissue is less vascular and more fibrous; giant cells are absent from these areas, but, between the interweaving bundles of collagen there are commonly seen groups of lipoid-filled macrophages which impart a yellow colour to the fresh tissue. As Jaffe and Lichtenstein (1942) remarked, these " foam cells " are not always present, but apparently increase in number as the lesion matures and as the cellularity of the connective tissue and the number of giant cells diminish. The appearances are not uniform throughout, and in a single lesion it is common to find cellular tissue towards the centre and more mature fibrous tissue at the periphery. According to Jaffe and Lichtenstein (1942) new bone formation is not observed within this aberrant tissue, though reactive sclerosis may be seen at the perimeter and normal callus develops at the site of a pathological fracture.

DISCUSSION

The radiographic and pathological features of non-osteogenic fibroma of bone are so characteristic that it is reasonable to suppose that this lesion is a distinct entity with a uniformly good prognosis. Jaffe and Lichtenstein (1942) based their original account on ten cases. Since then reports in the literature have suggested that non-osteogenic fibroma is not rare. Lichtenstein (1952) described it as " one of the more common benign connective tissue tumours of bone " and other authors appear to hold similar views of its frequency (Coley 1949, Coley and Higinbotham 1953). Hatcher (1945) reported a series of forty-five cases, seventeen of which were confirmed by biopsy. In five of these more than one bone was affected. We have been able to find only one case report in the recent British literature (Farrow 1954).

Two reasons may be given to explain the belated recognition of non-osteogenic fibroma of bone. Firstly, the increasing use of diagnostic radiography has no doubt brought to light many clinically silent cases which would previously have passed unnoticed. Secondly, cases have previously been interpreted, sometimes on the radiographic and sometimes on the pathological appearances, as examples or variants of: 1) localised osteitis fibrosa (Geschickter and Copeland 1949); 2) fibrous dysplasia of bone (Schlumberger 1946); 3) simple bone cyst (Geschickter and Copeland 1949); 4) osteoclastoma (giant-cell tumour of bone) (Kolodny 1927 and others); 5) solitary xanthoma, or a localised expression of skeletal lipoid granulomatosis (Hand-Schüller-Christian disease) (Burman and Sinberg 1938 and others); 6) fibrous osteomyelitis (Phemister 1929); and 7) endosteal fibrosarcoma (cited by Coley 1949).

The term localised osteitis fibrosa has been used to embrace so many different conditions that it is now almost meaningless and its use should be dropped. The monostotic form of fibrous dysplasia of bone is one of the disorders formerly included under the heading localised osteitis fibrosa, and Schlumberger (1946) held that non-osteogenic fibroma of bone should be regarded as a variant of fibrous dysplasia. Few authors, however, share this opinion and it may be noted that the skeletal distribution, histology and behaviour of the two conditions show important differences. Non-osteogenic fibroma occurring centrally may well be mistaken for a bone cyst on the radiographic appearances. The correct diagnosis can only be made with certainty in such cases by exploration and biopsy. Though there are some differences in the skeletal distribution of the two conditions, it is possible that they may be related to one another. Undue emphasis on the presence of osteoclasts on the one hand and foam cells on the other has led to mistaken diagnoses of osteoclastoma and xanthoma in cases of non-osteogenic

fibroma of bone cited by Jaffe and Lichtenstein (1942). These authors believed that the so-called "xanthic" or "healing" varieties of osteoclastoma are really instances of non-osteogenic fibroma of bone. The inconstancy of foam cells in non-osteogenic fibroma is sufficient ground for rejecting the use of the term "xanthoma" to describe the lesion. Phemister's (1929) conception of "chronic fibrous osteomyelitis" has long been refuted. Endosteal fibrosarcoma was the diagnosis made on histological grounds in a case of non-osteogenic fibroma mentioned by Coley (1949), in which unnecessary radiotherapy was given. The inclusion of cases of non-osteogenic fibroma of bone among other cases of a different nature makes tabulation of all the recorded cases difficult, but the distinctive radiographic and pathological features of this disease can be recognised in published illustrations which purport to show these other conditions. Thus Snapper (1949) gave an illustration of a typical radiograph of non-osteogenic fibroma under the heading of "lipoid granuloma of bone."

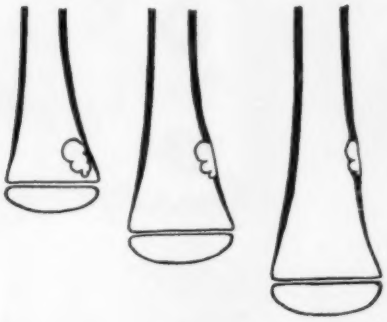

FIG. 27
Diagram to show the extrusion of a fibrous metaphysial defect in the remodelling process.

Several different views have been expressed on the nature of non-osteogenic fibroma of bone. Jaffe and Lichtenstein (1942) considered it to be a benign tumour arising from mature marrow connective tissue, and maintained that an essential feature of the growth was the complete absence of osseous metaplasia within the proliferating connective tissue. Lichtenstein (1952) upheld the view that this was a "genuine neoplasm," mentioning, in support, the rapid growth of "a proved instance of non-osteogenic fibroma." Hatcher (1945), on the other hand, contended that the lesion represents a local disturbance of bone growth originating at the epiphysial plate, and used the term "fibrous metaphysial defect" to describe it. Burrows (1950), among others, has adopted this name for the condition.

It may be argued that a benign tumour is, in any event, only a localised disturbance of growth of the tissue in which it arises. Certainly the difficulties of defining precisely what is meant by a benign tumour have become more and more apparent with the advance of knowledge. It is, however, widely accepted that true neoplastic growths seldom undergo spontaneous healing or dissolution, whereas such a course of events has been observed to take place in several instances of non-osteogenic fibroma of bone which have been followed by means of serial radiography, and there is other evidence to suggest that spontaneous disappearance of the lesion is a frequent outcome in this disease.

According to Hatcher (1945), "several modes of obliteration of the lesion have been observed." 1) Small lesions in the metaphyses of young children may undergo rapid "reparative ossification." 2) Lesions which persist longer and grow larger, as the epiphysial cartilage is carried away from them by longitudinal growth of the bone, tend to provoke sclerosis of the surrounding bone. If, as is commonly the case, the lesion is eccentric in position, remodelling and tubulation of the end of the growing bone will lead to a gradual extrusion of a small fibrous defect through the cortex and its final disappearance beneath the periosteum (Fig. 27). This process of extrusion is particularly likely to occur in sites where much remodelling of the end of the bone takes place during the growth period, such as the lower end of femur and upper end of tibia. When large defects are present in these situations, tubulation of the end of the growing bone is often imperfect. 3) In cases in which the lesion has not healed early, nor been completely extruded through the cortex of the bone, gradual obliteration of the focus may be observed by serial radiography (as in Case 5). The process may occupy several years, during the course of which the defect is seen to travel away from the epiphysis and its shadow to become less and less definite until finally it can no longer be distinguished. At

times the site of the obliterated defect is marked by a shadow of greater density than the surrounding bone; at other times the normal architecture of the bone appears to have been completely restored. Only an exceptional case fails to show evidence of spontaneous healing by the time longitudinal bone growth is complete.

The difficulty in elucidating the behaviour of non-osteogenic fibroma of bone is that the surgical intervention necessary to procure histological confirmation of the diagnosis is likely to alter the outcome. Yet it may be objected that cases which have been diagnosed on the radiographic appearances alone and which have not been confirmed histologically may not be instances of non-osteogenic fibroma of bone. This objection may be met by two arguments. Firstly, biopsy of lesions showing precisely similar radiographic changes regularly shows the histological picture of non-osteogenic fibroma of bone. Secondly, the rarity of non-osteogenic fibroma in radiographs of adult bones indicates that the lesion must frequently disappear spontaneously before adulthood. If these fibrous metaphysial defects undergo spontaneous obliteration presumably osseous metaplasia must take place within the connective tissue of the lesion, or else the fibrous tissue comprising the defect slowly shrinks and is absorbed, while reactive new bone grows in from the periphery. Unfortunately we have no precise knowledge of the sequence of changes in this healing process. If there is a stage when the connective tissue of the lesion itself becomes ossified, then the term " non-osteogenic " in the sense implied by Jaffe and Lichtenstein (1942), becomes inappropriate, apart from the question as to whether it is indeed a fibroma. Schlumberger (1946), whose views have been mentioned above, stated that metaplastic new bone formation was present in three out of twelve cases of non-osteogenic fibroma of bone filed under that diagnosis at the United States Army Institute in Washington. On the other hand, from the small number of cases that we have studied histologically, we have gained the impression that new bone does not arise by metaplasia within the connective tissue of the lesion itself. Obliteration of the focus seems to result rather from a gradual encroachment upon the shrinking fibrous tissue of reactive bone derived from the margin of the lesion and the septa dividing the loculi. Some, at least, of the newly formed bone is at first woven in type and is later replaced by lamellar bone.

The term " fibrous metaphysial defect " implies an origin in the metaphysis during the longitudinal growth phase of the bone and there is evidence to suggest that such defects may arise at any time during this period, though they most commonly do so at an early age. Sontag and Pyle (1941), who made a study of skeletal growth in 200 children by serial skeletal radiographs, found a surprisingly high incidence of " cyst-like areas " in the distal femoral metaphysis and particularly on the medial side. The " cysts " were often bilateral. None of these lesions gave rise to symptoms and none was biopsied, but their appearances, in the radiographs illustrated, are very similar to those of small fibrous metaphysial defects. The " cyst " was usually round, or oval, occasionally " sacculated " and sometimes surrounded by bone of increased density. The average age of the children at the time of appearance of these lesions was forty-six months and the lesion persisted for an average of twenty-nine months, gradually travelling away from the epiphysial cartilage with the growth of the bone. A few of the " cysts " were still present up to the age of nine or ten years, but all eventually disappeared spontaneously. It is possible to make a rough estimate of the duration of a fibrous defect by the distance which separates it from the epiphysial plate, if it be assumed that this represents its point of origin. A remarkable case was recorded by Ponseti and Friedman (1949) of a boy who first came under observation at the age of two years on account of scoliosis. The initial radiographs included the humeri, in one of which a small metaphysial defect was noted close to the upper epiphysis. By the age of five this had grown larger, travelled away from the epiphysis and had become indistinct, but a second defect was already apparent in the same region of the metaphysis where the first had begun. This followed a similar course till it disappeared by the age of nine years, when a third and final lesion developed. This, in turn, travelled down the shaft and had disappeared by the age of thirteen.

These observations on young children suggest that fibrous metaphysial defects may arise in early life, but heal readily at this period. The cases which have been reported as non-osteogenic fibroma of bone come mostly from older children, though it seems probable that these are not fundamentally different from the lesions observed radiographically in infants and young children. As already remarked, non-osteogenic fibroma of bone is uncommon in adults. We have seen the radiograph and biopsy section of an adult of forty-two (Jaffe 1953) in whom the lower end of the radius was expanded by a bulky tumour which simulated an osteoclastoma, though the histological appearances were typical of non-osteogenic fibroma of bone. In view of the juxta-articular situation of this defect it is reasonable to assume that it must have arisen at or after the time of skeletal maturation; in this case the bone showed no tendency to undergo spontaneous healing.

The etiology of fibrous metaphysial defects is unknown. It is evident from their distribution that they arise most commonly in connection with the most actively growing epiphysial cartilages in the body, and the eccentric situation of most lesions suggests an origin from the peripheral part of the epiphysial plate. Hatcher (1945) found concomitant epiphysial disorders in no less than fourteen of his forty-five cases. These comprised eight cases of Osgood-Schlatter's disease, four cases of osteochondritis dissecans and two cases of " osteochondritis " of the patella. He postulated a vascular disturbance as the underlying cause of both the metaphysial and epiphysial disorders. In one of our ten cases (Case 4) a metaphysial defect in the lower femur was associated with Osgood-Schlatter's disease on the same side. Among other recorded cases, only that of Burman and Sinberg (1938) was accompanied by Osgood-Schlatter's disease. The lesion was an incidental radiographic finding in the lower end of the femur of a boy of twelve whose complaint was pain over the tibial tuberosity. We have examined the radiographs of twenty consecutive cases of Osgood-Schlatter's disease and not found any fibrous metaphysial defect. The case of Ponseti and Friedman (1949) quoted above, in which multiple defects appeared to arise from the same point in the epiphysial plate, might be taken to favour some such mechanism as a local vascular disturbance of the growth cartilage. Certain of the histological features are indicative of repeated haemorrhage (extravasated red cells, haemosiderin pigment and lipoid-filled macrophages), and the tissue reaction has these features in common with the " sclerosing angioma " of soft tissues. It is perfectly clear that the lesion is not merely an organising haematoma. In spite of the rapid enlargement of such lesions, which has occasionally been observed, we do not believe that there is sufficient evidence for regarding the disorder as a neoplasm, and we suggest that the term " fibrous metaphysial defect " is therefore more appropriate than " non-osteogenic fibroma of bone." Furthermore, rare cases of non-bone-forming, endosteal fibromas have been described (Cappell 1935, Geschickter and Copeland 1949) and these have little in common with the lesion under discussion. There is no recorded instance of malignant change supervening in a fibrous metaphysial defect. Several authors, however, have reported the incidental discovery of these defects in radiographs which also show osteogenic sarcomata (Katz and Marek 1950, Lichtenstein 1952).

From the above observations it follows that no specific treatment is required if the radiographic appearances are sufficiently characteristic to establish the diagnosis, and if there are no complications. Where doubt exists, where the lesion involves the whole width of the bone, or where there is a pathological fracture, exploration and packing with bone chips may be necessary and should expedite healing.

SUMMARY

1. Ten cases are recorded of the entity known as non-osteogenic fibroma of bone.
2. We believe the evidence is in favour of the condition's being a localised disturbance of bone growth (metaphysial fibrous defect) rather than a true neoplasm.
3. The disorder usually pursues a symptomless course and in many instances the lesion disappears spontaneously.

We are most grateful to Mr S. A. Jenkins and Mr G. P. Arden for their active help in sending details of their cases, to Mr Charles Gray for his encouragement and permission to use his cases, to Dr H. A. Sissons for his helpful comments on the manuscript of this paper and to Mr J. S. Sheppard and Miss D. L. M. Bannister for the photomicrographs, radiographic prints and photographs.

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SPONDYLOLISTHESIS OF THE CERVICAL SPINE

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Spondylolisthesis is an abnormality that is often seen in the lumbar spine. It is rare to find it in the cervical region although congenital abnormalities of the cervical spine are often observed. A case of true cervical spondylolisthesis was recorded by Perlman and Hawes (1951), but this is the only previously recorded case so far as is known. Another case is presented here.

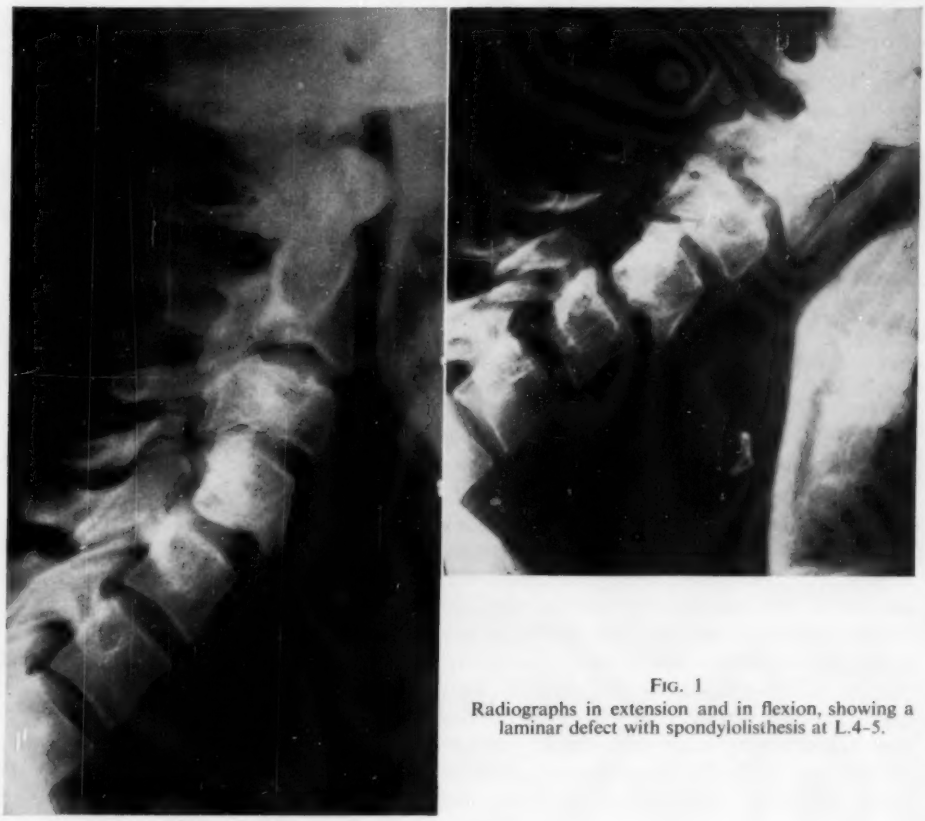

FIG. 1
Radiographs in extension and in flexion, showing a laminar defect with spondylolisthesis at L.4–5.

CASE REPORT

A bricklayer's labourer, aged twenty-five, gave a history that four days previously he had awakened to find his neck stiff and painful and that he could not move it towards the left. This state of affairs had persisted. He had not injured his neck nor had he before suffered pain in it. In his job he had to carry a hod of bricks on his left shoulder and he had been doing this without trouble on the day before his symptoms appeared. He had not suffered pain or tingling in the upper limbs.

On examination, there was a right torticollis. Neck flexion was full and painless. Lateral flexion was restricted and painful. Extension was painful. There were no abnormal neurological signs in the upper extremities. Radiographs revealed a bilateral laminar defect of the fourth cervical vertebra (Fig. 1).

The neck was gently manipulated, and he was put at rest in bed and instructed in neck extension exercises. Four days later he had full movement and no pain. Radiographs in flexion showed some instability of C.4 on C.5. His job involved the carrying of heavy weights

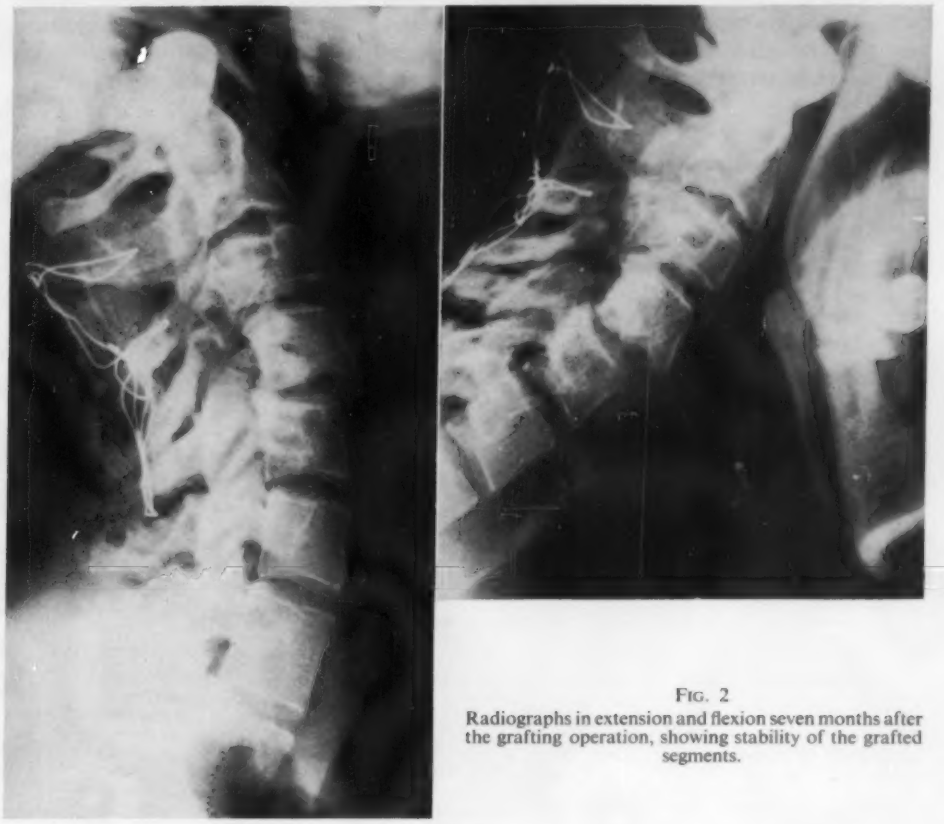

FIG. 2
Radiographs in extension and flexion seven months after the grafting operation, showing stability of the grafted segments.

on the shoulder and imposed strain on the neck; so local fusion by bone grafting was performed later. Iliac bone was used and fixed in position with stainless steel wire. The second cervical vertebra was included because the spinous process of C.3 was small. At operation the spinous process and laminae of C.4 were found to be completely mobile. A plaster collar was applied for three months. Flexion radiographs after its removal showed that the cervical spine was stable (Fig. 2). He is now back at work on full duty.

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PRIMARY RUPTURE OF BRACHIAL ARTERY AND MEDIAN NERVE IN SUPRACONDYLAR FRACTURE OF THE HUMERUS

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This paper records a case of supracondylar fracture of the humerus in which the brachial artery was completely severed and the median nerve interposed between the fragments, preventing reduction. Reference will also be made to another case, explored three months after injury, in which there had been complete primary rupture of the median nerve.

CASE REPORT

A boy aged seven years was admitted to hospital shortly after falling out of a tree. He was found to have closed fractures of both bones of the left forearm in their lowest thirds,

FIG. 1
Radiographs before reduction.

and a severely displaced left supracondylar fracture in which the upper fragment had at one point penetrated the skin of the grossly swollen antecubital region. There was no pulse at the wrist, but the colour of the hand was good and nerve function normal.

A cautious attempt was made to reduce the fracture. This was unsuccessful; the upper fragment could not be moved back from its subcutaneous position. Accordingly, the small punctum was excised and the antecubital fossa opened anteriorly. The median nerve was found caught and kinked between the fragments, in contact with the jagged edges of the upper. Of the brachial artery there was at first no sign, but after a search its proximal severed end was found and tied. The distal end had disappeared and was not sought further. The expanded lower end of the upper fragment had passed completely through the brachialis, severing the greater part of that muscle. The biceps tendon was intact, although the lacertus fibrosus

had been torn. The median nerve was freed and the fracture was reduced under direct vision. Post-operative progress was uncomplicated. The circulation in the hand was never a source of anxiety, though a palpable radial pulse did not return for two months. There was slight partial sensory impairment in the fingers, and motor weakness of median distribution, both of which recovered after about eight weeks. Eight months after the injury there was full

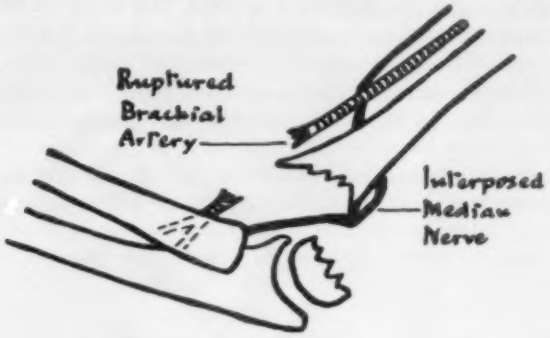

FIG. 2
Diagram showing the findings at operation.

extension of the elbow, flexion to twenty degrees beyond the right angle, and a good range of rotation. Figures 1 and 2 illustrate this case.

OTHER CASES

Rupture of the brachial artery in supracondylar fracture is rare. Three cases were published in 1951 by Spear and Janes of the Mayo Clinic, and since this paper was written one more has appeared, but without clinical history, in a ten-year analysis of the neurovascular complications of supracondylar fracture from the same centre (Lipscomb and Burleson 1955).

TABLE I

FOUR CASES OF RUPTURE OF THE BRACHIAL ARTERY

Age	Injury	Simple or compound	Treatment of artery	Co-existing fractures	Muscle damage
6	Fall from tree	Simple	Nil	Both forearm bones	Not recorded
14	Car accident	Compound postero-medially	Both ends tied	Nil	Biceps and brachialis ruptured
12	Fall from bicycle	Compound anteriorly	Both ends tied	Both forearm bones (compound)	Biceps and brachialis ruptured
6	Fall from tree	Compound (small anterior puncture)	Proximal end tied	Both forearm bones	Brachialis and lacertus fibrosus ruptured

There are no others, although Spear and Janes record their impression that this injury is more common than the scarcity of literature would suggest.

It is interesting to compare the case under discussion with the three previous instances of arterial rupture whose histories are reported. All four were fractures of boys' left arms. Three were compound, though our own case was only barely so. The severity of the fracturing force is illustrated by the fact that three, including the simple fracture, were accompanied by fractures of both forearm bones on the same side. In no case was there any serious circulatory embarrassment or nerve damage. In Table I the four cases are listed in the order in which they were described, ours being the last.

It is worth noting that arterial rupture seems rather more common in association with dislocation of the elbow. Twenty-four such cases with main vessel rupture have been published, eighteen involving the brachial artery. Fifteen of these eighteen were included in a review of the literature going back to 1880 (Eliason and Brown 1937); two were published in Britain (Henderson and Robertson 1952); and the other by Spear and Janes along with the three in supracondylar fractures already discussed.

Rupture of the median nerve in supracondylar fracture is even rarer than arterial rupture. Apparently only a single case has been reported—by Bristow (1923), who explored the elbow three months after injury on account of ischaemic contracture. He considered at operation that the nerve had been divided " not at the time of the accident, but rather at the subsequent manipulation."

A recent case, for which I am indebted to Mr J. S. Ellis and the staff of the Treloar Hospital, is thus of some interest. This occurred in an eight years old girl, operated upon three months

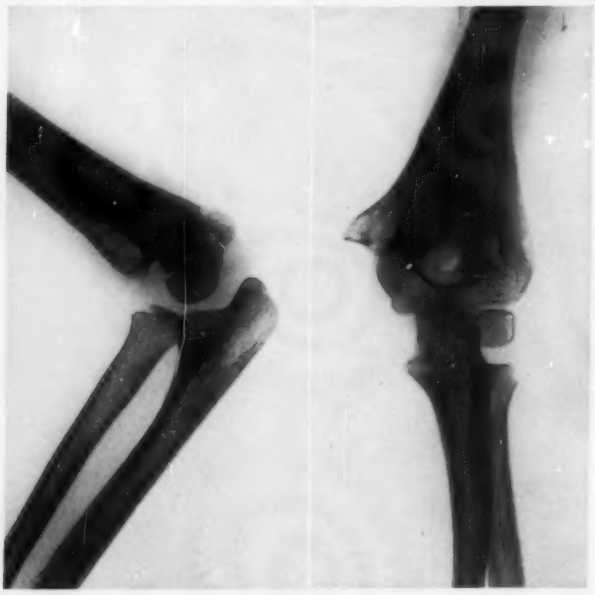

FIG. 3
Radiographs three months after a supracondylar fracture complicated by complete median nerve palsy.

after supracondylar fracture, on account of a persistent median palsy. On admission there was malunion, with postero-lateral displacement of the distal fragment and a tender bony spur on the medial side (Fig. 3). At operation the spur was exposed and a fibrous cord found crossing it superficially. This cord connected a neuroma some two inches proximal to the spur with the distal stump of the median nerve about one inch below the spur. Both spur and cord were excised, and end-to-end suture of the median nerve was performed. After operation there was no detectable motor or sensory change, and this fact, coupled with the operative findings, entitles the case to be considered one of complete primary nerve division. It is similar to Bristow's case in that it came to operation three months after injury, and differs in being uncomplicated by ischaemic contracture.

The remarkable capacity of the median nerve to remain unscathed in the presence of severe damage to its immediate neighbourhood is also evident in posterior dislocation of the

elbow. Eliason and Brown (1937) could find only one proven case of median nerve division—reported by Holl in 1880—and this despite the occurrence among their cases of " rupture in different combinations of nearly every tendon or muscle anterior to the elbow."

Partial injuries and irritations of the neurovascular bundle are, of course, more common and in the case of arteries more dangerous in their effects. Even so, it is exceptional to find damage of any kind to the median nerve in supracondylar fracture. Siris (1939), reviewing 330 cases, 244 of which had bad posterior displacement and twelve of which were compound, had not a single case of median nerve damage. Watson-Jones (1930) had no case in a series of eighty-seven such fractures, although he states elsewhere (Watson-Jones 1943) that 15 per cent of these fractures are accompanied by median or ulnar nerve contusion. Platt (1928) described four cases of injury to the nerve, all with full recovery save one occurring in conjunction with Volkmann's ischaemic contracture. Lipscomb and Burleson (1955) recorded seventeen nerve injuries in 108 cases of extension-type supracondylar fracture. Two patients had involvement of more than one nerve, so that altogether there were eleven radial, seven median and two ulnar lesions among the seventeen patients. All were partial injuries with complete recovery, save one which had persistent intrinsic muscle weakness in the hand. Four were explored surgically, but the operation in each case consisted of neurolysis of an intact nerve. In the same series there were—apart from complete ruptures—nine instances of involvement of the brachial artery including one of ischaemic contracture.

ANATOMICAL CONSIDERATIONS

In a displaced extension-type supracondylar fracture the sharp upper fragment first embeds itself in the brachialis. If the force is sufficiently severe, the muscle will fail to arrest the forward-driven shaft which will then enter the antecubital fossa and, after encountering the biceps tendon, usually pass medially among the neurovascular structures. Operative experience in both supracondylar fracture and dislocation of the elbow confirms this observation for which there are two anatomical reasons. First, the position and direction of the biceps tendon, which at fracture level is a smooth cord lying just lateral to the anterior border of the humerus—that is, on the steeply inclined antero-lateral surface of that bone, at the top of the slope, and with its direction towards an insertion still more laterally placed. Secondly, the mechanism and direction of the fracturing force which, with the elbow extended, will tend to direct the humerus medially in relation to the forearm, because of the carrying angle.

I believe that these factors will usually govern the initial displacement and the relations of the upper fragment to the soft tissues, irrespective of whether the distal fragment later becomes medially or laterally displaced. Siris (1939) found medial and lateral displacement of the distal fragment in equal frequency, and it is not uncommon to find the position change from one to the other during the course of ordinary manipulative reduction. But once the proximal fragment has completely penetrated the brachialis and come to lie among the neurovascular structures, movement of the distal fragment to one side or the other—though it may still occur—can do nothing to relieve the danger of the position. In the case described the humerus passed between the biceps and the median nerve, severing the intervening artery and lacertus fibrosus, and finally came to lie under the skin with the nerve behind it in the path of reduction.

The presence, however, of a palpable subcutaneous spike of bone in the antecubital fossa does not necessarily mean that this dangerous anatomical situation has been reached, for the whole expanded end of the broken shaft may not have passed through the brachialis; in which case, when reducing forces are applied, the ensheathing muscle fibres will guide it safely backwards as the scabbard guides a homing sword. That reduction does in fact generally occur thus is affirmed by Charnley (1950), who considers that temporary incarceration of neurovascular structures is a common accompaniment of severe displacement, though in

most cases unlikely to cause permanent damage provided the surgeon releases them by applying traction before attempting to flex the elbow. Radiographs, with good soft-tissue visualisation, may sometimes help in deciding this point; in the films of the case described it can be seen that the entire lower end of the shaft has passed through the muscle shadow to occupy a subcutaneous position.

DANGERS OF MANIPULATION

The necessity for accurate reduction on the one hand and the dangers of manipulation on the other have been stressed by many authorities. Bristow's opinion that his case of ruptured median nerve was caused by the manipulation rather than the fracture is balanced by Platt's remark that "prevention of median nerve involvement is largely a problem concerned with the efficient reduction of supracondylar fractures." Again, Fairbank (1936) stated that in his experience Volkmann's ischaemic contracture was excessively rare without unreduced gross displacement, and Griffiths (1940) on the same subject wrote that " the first symptom of the arterial block dates more often from a manipulation than from the fracture itself." Are there, then, circumstances in which the dangers of closed manipulation are so great that open reduction should be advised?

OPEN REDUCTION

Before attempting an answer it is worth recalling the axiom propounded by Fagge (1936) that if the deformity is really reduced, the fragments impinge so firmly that movement at the elbow takes place before movement at the fracture. From it he inferred that one should never depend upon a particular position for the maintenance of alignment, and it was his practice, if he could not secure reduction with the elbow at a right angle, to perform open operation.

It was stated by Charnley (1950) that, using a technique of manipulation based on anatomical knowledge of the fracture, there should never be any necessity for open reduction, except for nerve or arterial complications. This is a sound working guide and nearly, but I think not quite, always true, especially where several previous manipulations have been tried. In such circumstances a careful open operation may well be preferred to continuous traction or mechanical traction, as advocated by Böhler (1935), Madsen (1955) and others.

Absence of the radial pulse, it is generally recognised, can occur without any true impairment of circulation in the hand, or fear of Volkmann's contracture. Such absence, which may persist for days or weeks, despite good reduction and an otherwise normal hand, is not therefore of itself an indication for operation. If, however, it is accompanied by pale, flexed fingers, painful when passively extended, the signs of early ischaemic paralysis are present and demand immediate surgical intervention.

Girdlestone (1936) defined his objectives at operation as threefold: to evacuate the haematoma, to safeguard the neurovascular bundle and to obtain accurate reduction. With these in mind, I have ventured to enumerate the criteria that demand operation as follows. 1) Compound fractures; 2) antecubital tension, extreme in degree or unrelieved by reduction; 3) ischaemia of the hand or suspicion of Volkmann's contracture, especially if persisting after reduction; 4) uncertainty about the position, especially suspected anteposition, of the upper fragment relative to the neurovascular structures, depending upon whether penetration of brachialis is complete or incomplete; 5) nerve palsy associated with incomplete reduction (Platt 1928); 6) failure of closed reduction under full general anaesthesia (Girdlestone 1936); 7) failure to secure a stable closed reduction with the elbow at a right angle (Fagge 1936).

SUMMARY

1. A case, believed to be the fifth on record, of supracondylar fracture with rupture of the brachial artery is described.

2. The relative immunity of the median nerve in these injuries is discussed, with brief reference

to a recent case of complete rupture. Only a single previous report of this complication could be found.

3. It is suggested that these injuries are less uncommon than the number reported would indicate.

4. The anatomy of severe displacement is discussed, with special reference to the role of the brachialis.

5. The danger of closed reduction when the relationship of the upper fragment to the neurovascular bundle is in doubt is stressed.

6. The indications for open reduction are given.

I am very much indebted to Mr James Ellis and Mr F. Wilson Harlow for their helpful and kindly criticism of this paper.

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ACUTE ATROPHIC CHARCOT'S DISEASE OF BOTH HIPS

Report of a Case

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Acute atrophic Charcot's disease of the hip is not a common condition, and cases in which it is possible to demonstrate radiographically the rapidity of the destructive process are extremely rare.

Chitty (1939) reported a case in which there was complete loss of one femoral head from Charcot's disease five months after the patient had sustained a fracture of the pelvis, when both hips were seen to be normal radiographically.

The case now reported is one of Charcot's disease of both hips. The femoral head on one side is shown to have been completely absorbed in less than five months, with no history of precipitating injury.

CASE REPORT

The patient, a woman of sixty-four, gave a history that while walking in the house ten days before admission to hospital her left leg suddenly gave way and she fell to the ground, since when she had been unable to walk. Swelling of the whole leg commenced two days later, and gradually increased.

At no time was there pain in the leg, but there had been " pins and needles " in both legs for several years.

On examination, the whole of the left lower limb was swollen and oedematous, with most swelling in the thigh, where there was some reddening of the skin. The peripheral pulses were all present. The sense of touch in both legs was impaired, and there was loss of all pain sensation and of vibration sense. Knee and ankle jerks were absent in both legs. Examination of the eyes showed Argyll-Robertson pupils.

There was gross instability of the left hip, but movements were painless.

Radiographs showed complete destruction of the head of the left femur, with some calcification in the surrounding soft tissues (Fig. 1). The Wassermann reaction was positive.

A diagnosis of acute atrophic Charcot's disease was made. The swelling slowly subsided with rest in bed and the patient was discharged from the ward.

When she was seen three weeks later the swelling had completely disappeared and she was managing to walk with a stick, although she had a severe limp.

The patient was readmitted three months later with the history that three weeks previously her right leg had suddenly let her down, and she had been unable to walk since. At the same time the leg began to swell, and this swelling steadily increased.

On examination, the whole right lower limb was swollen and oedematous. The swelling had an inflammatory appearance, especially in the thigh, where some indurated areas were palpable and the skin was reddened (Fig. 2).

There was no change in the neurological signs, as compared with the previous examination.

The right hip now showed the same gross instability as the left, and again there was an abnormal range of painless movement.

Radiographs showed that the head of the right femur had been completely absorbed, and soft-tissue calcification was present around this joint also (Fig. 3).

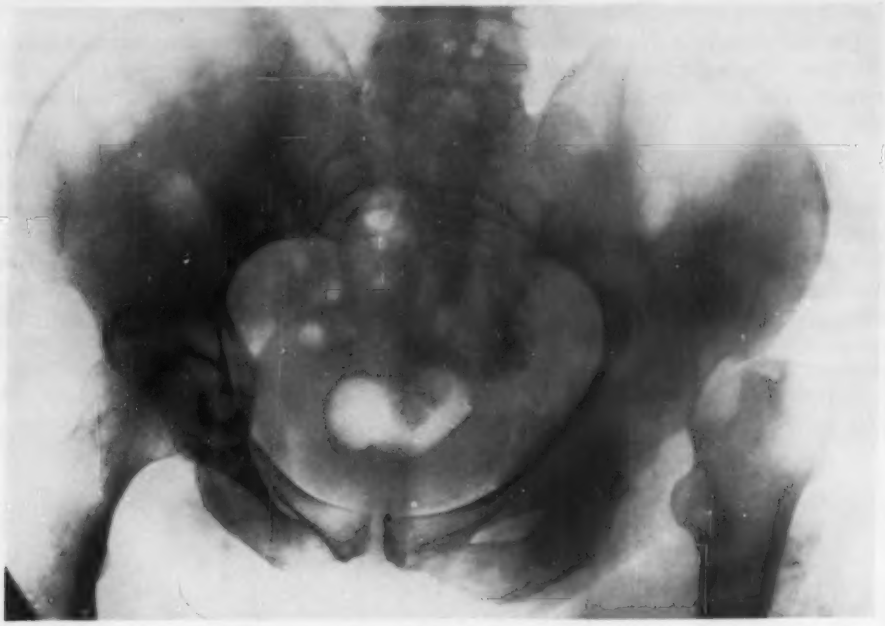

FIG. 1
Initial radiograph. Charcot's disease of left hip. Right hip unaffected.

FIG. 2

FIG. 3

Figure 2—Marked oedema of right lower limb complicating Charcot's disease of the hip. Figure 3—Radiograph of the right hip taken five months after that shown in Figure 1, showing destruction of the head and neck of the femur.

DISCUSSION

The radiographs in this case show the typical appearances of atrophic Charcot's disease of the hips. The right femoral head was demonstrated to be normal at the time of the first admission, and less than five months later had been completely absorbed. There was no history of injury.

Another interesting feature of the case is the massive oedema of the whole limb. There was a similar swelling, confined to the thigh, in Chitty's case, and he suggested that the oedema around the joint was due to rupture of the joint capsule and liberation of a large quantity of synovial fluid into the soft tissues.

The history in the present case is consistent with this view, because in each leg the swelling commenced soon after the collapse of the affected joint, at which time the capsule probably ruptured. The local oedema around the joint may then interfere with the lymphatic and venous return from the leg, producing oedema of the whole leg and foot.

I wish to thank Mr F. W. Holdsworth for permission to publish this case.

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ELECTROLYSIS AND STAINLESS STEELS IN BONE

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There is still much mystery in the loosening of metal screws inserted in bone. Why do some loosen while others remain so fixed that they will break rather than rotate when the surgeon's screwdriver tries to remove them? There is, of course, no single answer to this question. Some screws loosen because they are inserted badly; others through faults in their metals; some from sepsis and some from other causes. In this paper we will discuss the part played by electrolysis in this loosening, a factor which has been brought to our attention once again by the work of Bowden, Williamson and Laing (1954). We do not propose to discuss the non-ferrous alloys (Vitallium, etc.) but to confine our attention to the more commonly used stainless steels.

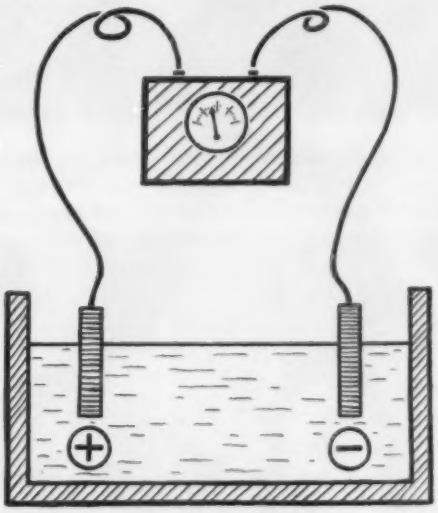

FIG. 1
The simple voltaic cell—differing metals immersed in an aqueous electrolyte.

THE VOLTAIC CELL

If two different metals or alloys of different chemical composition are in electrical contact in the presence of an aqueous electrolyte, a voltaic cell is set up (Fig. 1), and an electric current will flow from one piece of metal, through the electrolyte, to the other. As regards the flow of positive electricity, the piece of metal from which the current flows into the electrolyte is designated the anode, and suffers electrolytic dissolution or corrosion. The other piece of metal, into which the current flows from the electrolyte, is designated the cathode, and receives a deposition of hydrogen ions or metal ions. If oxygen is present on the cathode it may combine with the discharged hydrogen ions to form water.

If the anode is a screw inserted into bone, living or dead, corrosion will cause it to loosen. This is due partly to the loss of metal from its surface, but much more to the superficial disappearance of the bone with which it is in contact. The loss of bone may be due to its

active involvement in the electrolytic process, but it is more likely that the bone is attacked by the products of electrolysis as a purely chemical phenomenon. If these changes occur in living tissue the gap left by loss of bone is filled with fibrous tissue in which small metallic particles are embedded (Fig. 2). This fibrous tissue, though tough, has not the strength of

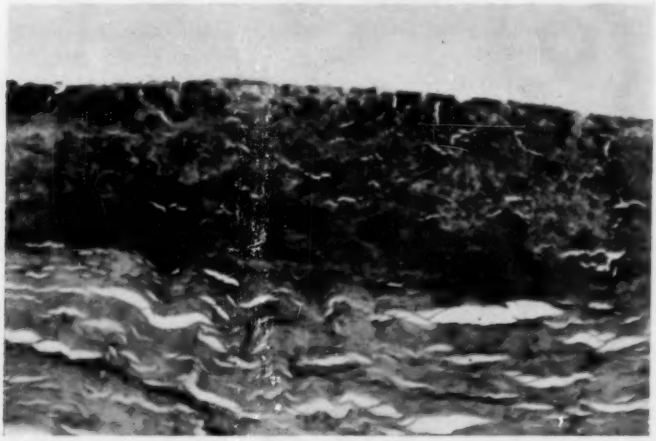

FIG. 2
Metallic particles in fibrous tissue near a corroded screw. (Prussian blue, ×110.)

bone, and if the screw is under considerable mechanical strain will fail to hold it. Once any movement of the screw is possible the " play " is gradually increased mechanically, as occurs when one removes a nail from a piece of wood by repeatedly bending it in opposite directions. The electrolytic bony dissolution produces a shallow, conical defect which can be seen in a radiograph around one or other extremity of the screw (Fig. 3). Movement produces a

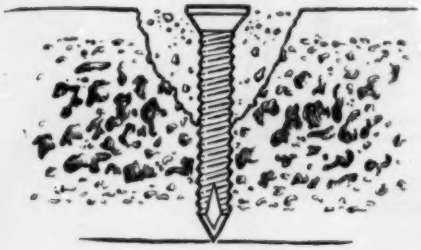

FIG. 3

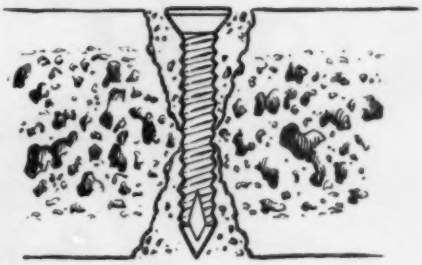

FIG. 4

Figure 3—Pattern of bony erosion due to electrolysis. Figure 4—Pattern of bony erosion due to movement of the screw.

narrower conical defect visible at one or both ends (Fig. 4). Such movement predisposes to the fracture of the screw at the fulcrum when the bone it is securing is " shock-loaded " by a sudden strain or fall. This is more likely to occur if the screw is also locally corroded.

If the electrolytic process is speeded up by passing an electric current from a battery to two screws inserted into dead bone and immersed in an electrolyte such as normal saline or plasma, changes can be produced identical with those occurring *in vivo*. Figures 5 to 7 show the result of such an experiment. The stainless steel screw shows extensive destruction, and the surrounding bone is removed. It is interesting to note that bone is here being removed

by an electro-chemical process and not by osteoclasis. Whether or not this can occur *in vivo* is unknown, but the similarity of radiological appearance in both cases suggests that it is possible.

The amount of corrosive attack depends on the amount of current per unit of time

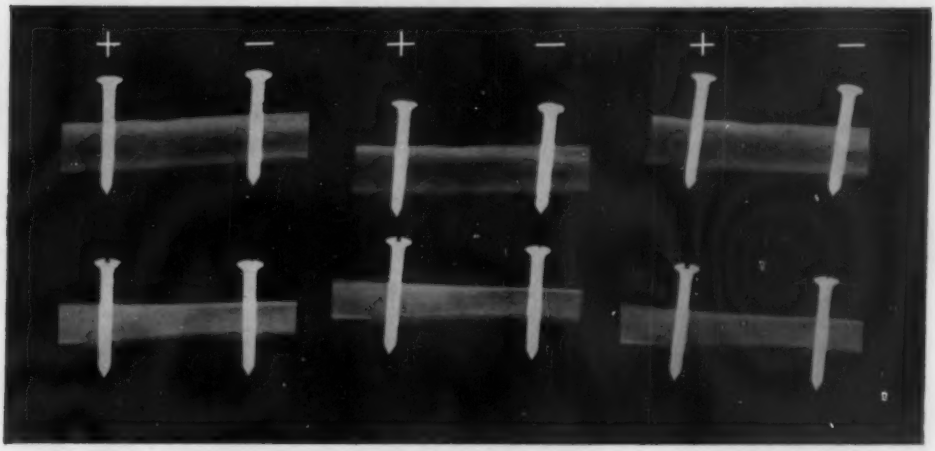

FIG. 5

Radiographic appearances at 0, 20 and 40 hours after passage of 10 milliamp current, with bone and screws immersed in bath of normal saline. (Control below.)

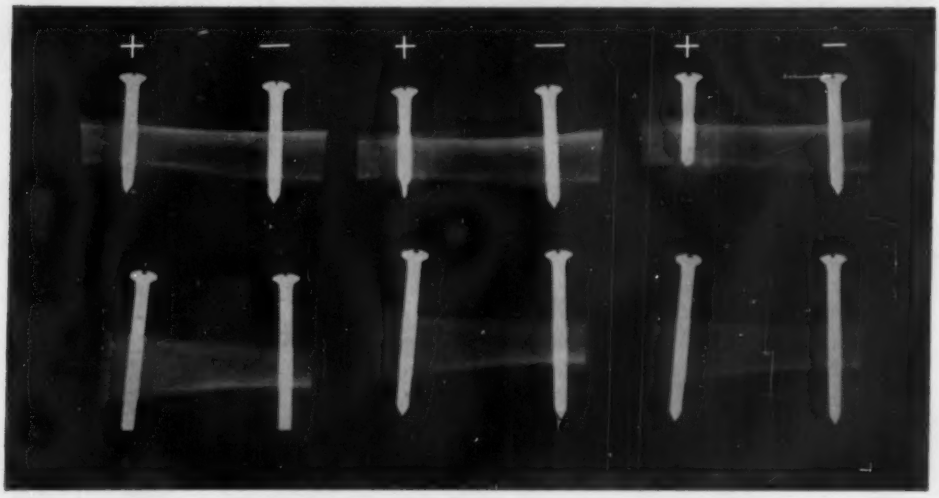

FIG. 6

Similar experiment with bone mmersed in human plasma. Marked corrosion of stainless steel screw.

passing through the voltaic circuit and hence upon the electrical conductivity of the circuit. In isolated screws in bone (as in an onlay graft) the current flowing depends upon the conductivity of the tissue fluids between them. If the screws are joined electrically by a plate between them the conductivity of the circuit is increased, and consequently more corrosion can be expected to occur.

It is precisely to prevent the above phenomena that surgeons use stainless steel materials in the body, as these, which have been presumed to be of uniform composition, are regarded (Venable and Stuck 1947) as less electrically active. Yet screws of stainless steel do loosen in bone. Can such loosening be due to electrolysis, and, if so, how important is this factor as a cause of non-union of fractures treated by internal fixation with screws and plates of stainless steel?

FACTORS INFLUENCING THE CORROSION OF STAINLESS STEEL SCREWS

There are five important factors that may lead to corrosion of stainless steels and to loosening of screws composed of it. They are 1) chemical composition of components; 2) surface abrasion; 3) cold welding; 4) mechanical distortion; 5) differential oxygenation.

Chemical composition of components—All " stainless steels " are not of the same chemical composition; there are, indeed, many different types. One of these, designated 18/8, contains approximately 18 per cent of chromium and 8 per cent of nickel. The addition of about 2 per cent of molybdenum to such a steel gives an increased resistance to corrosion in the presence of chloride ions and organic acids—as occurs in the tissues of the body. Venable and Stuck have stated the composition limits of a satisfactory stainless steel for use in the body as follows: chromium 17 to 20 per cent; nickel 10 to 14 per cent; molybdenum 2 to 4 per cent; sulphur 0·03 per cent maximum; iron to 100 per cent. Such a material is designated Number 317 by the American Iron and Steel Institute, and is referred to by Venable and Stuck as 18/8 S.Mo.

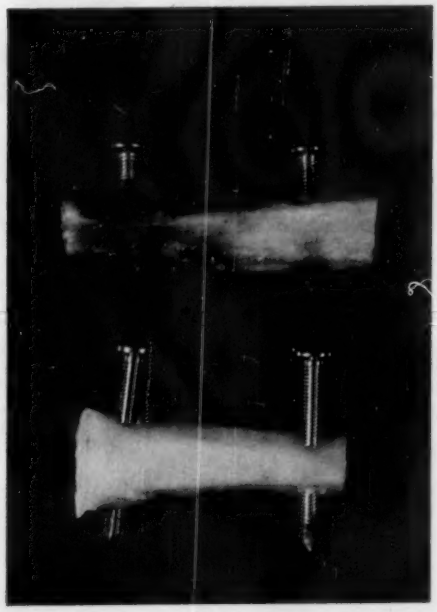

FIG. 7
Photograph of the screws shown in Figure 6.

The screws made in Great Britain for orthopaedic surgery do not always conform to these standards. Examination of a nut, screw and washer of approximately 4.BA size of the so-called Sherman pattern showed: chromium 20·5 per cent; nickel 9·5 per cent; sulphur more than 1 per cent; molybdenum nil. Spectrographic examination indicated that nut, screw and washer were all made from material of an identical chemical composition, although, as will be shown later, the three components were not in the same metallurgical condition. The absence of molybdenum and the excess of sulphur (more than three times the permitted maximum) should be noted. It appears, therefore, that the stainless steel supplied for orthopaedic use in Great Britain can differ from that recommended by Venable and Stuck.

Metallographic examination of a polished section of one of these screws showed the presence of many small isolated crystals embedded in the mass of the iron-chromium-nickel alloy (Fig. 8). The crystals consist of metallic sulphides and are hard and brittle. The presence of such crystals of hard compound embedded in the relatively soft iron-chromium-nickel alloy makes the cutting of screw threads much easier, and such a material is called " free cutting." Since these crystals are uniformly distributed throughout the alloy, a voltaic cell may be set up between the crystalline compound and the mass of alloy itself, if the material is in contact with an electrolyte, as they act as two electro-chemically different materials. " Free cutting " stainless steel would be expected to show inferior corrosion resistance to a

steel consisting of one type of material. It is noteworthy that we did not find these crystals in a different screw of the " wood screw " type.

The effect of surface abrasion—The resistance of stainless steel to corrosion depends upon the presence of a thin, invisible but continuous film of chromium oxide which covers the surface and isolates the metal from a corrosive electrolyte. Screws and plates for orthopaedic use are usually electropolished to produce a smooth surface, and then " passivated " in nitric acid to increase the effectiveness of the oxide film. This film is some 10^{-5} millimetres thick and can be easily destroyed by mechanical abrasion. Such abrasion readily occurs at the edge of a hole in a plate by contact with a rotating drill; on the head of the screw by mechanical damage from a screwdriver; at the points of contact between a screw and an edge of the plate, and especially when the male and female threaded portions of a nut and bolt are fastened tightly together. Such mechanical abrasion removes the insulating film, and electrolytic corrosion can begin.

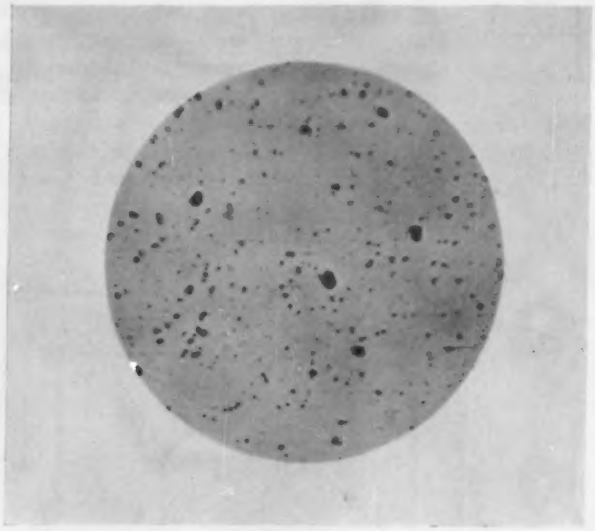

FIG. 8
Polished section of screw showing isolated crystals of metallic sulphides in iron-chromium-nickel alloy.

The " cold weld " effect—Bowden, Williamson and Laing (1954) demonstrated another mechanism by which electrolysis may be produced around and on screws and plates of stainless steel. When a screwdriver is placed in firm contact with a screw, as it must be when the screw is driven home, an actual transfer of metal occurs from the screwdriver to the screw, and vice versa. Another similar transfer occurs when a clamp is applied tightly to a plate, or when any two metals are forced together and later separated. Microscopically the surfaces of these articles are not smooth, but irregular. When two irregular surfaces are clamped together, " high spots " occur where salient points meet, and the pressure at these points produces an actual welding together of the metals (a " cold weld "). When the metal surfaces are later separated, they may fracture apart on either side of some or all of these points, thus transferring metal from the screwdriver or clamp to the screw or plate (Fig. 9). There is no doubt that this can occur. Bowden, Williamson and Laing (1954) showed by radiochemical studies that up to 65μ-grammes of anodic material can be transferred from a screwdriver to a screw. The importance of this is doubtful, but its existence is certain.

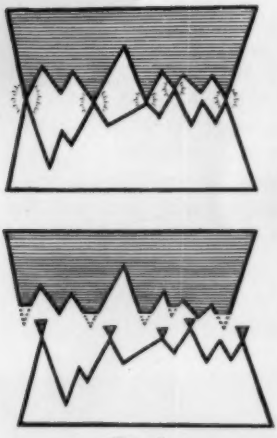

FIG. 9
"Cold weld" effect.

The effect of mechanical distortion—If a piece of almost any common metal is distorted mechanically the distorted portion tends to become anodic with respect to the undistorted portion. This is due to the greater ease with which atoms from an irregular (distorted) lattice arrangement can dissolve than can those of a regular (undistorted) lattice. Such distortion cannot fail to occur during the process of cutting a screw thread, since the process is essentially one of tearing a furrow in a piece of metal.

In stainless steels of the 18/8 type mechanical distortion may also actually change the type of crystal lattice on which the metallic atoms are situated. The 18/8 type of steel exists as a face-centred cubic crystal, an arrangement as shown in Figure 10. This crystal is non-magnetic and is called "austenite." Mechanical distortion may change this non-magnetic austenite to a magnetic crystal type termed "martensite." The amount of the magnetic modification increases with the degree of mechanical distortion. This magnetic modification of the iron-chromium-nickel alloy ("martensite") has a crystal structure in which the atoms are arranged in a body-centred cubic pattern (Fig. 11).

When this effect is related to the nut, screw and washer already mentioned (free cutting steel containing 20·5 per cent chromium and 9·5 per cent nickel) the washer showed no magnetic response to the hand magnet, the nut showed a weak magnetic response if the magnet

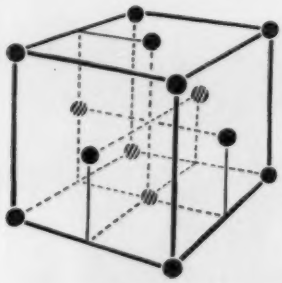

FIG. 10
Face-centred cubic crystal.

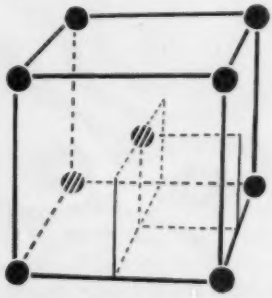

FIG. 11
Body-centred cubic crystal.

was placed near to the screw threaded hole, and the screw itself showed a strong magnetic response at the threaded portion, though not at the head.

This illustrates how mechanical distortion in the process of cutting a screw thread may put chemically similar articles into different metallurgical conditions though originally they were fashioned from the same block of metal. The electro-chemical effect is the same as though two different alloys were present, and a voltaic cell can thus be produced. Mechanical distortion may also occur of course during the insertion of a screw, with similar changes.

The susceptibility of 18/8 type of stainless steel to this effect depends upon the proportion of nickel to chromium. The role of chromium is to provide resistance to corrosion by the formation of a continuous and coherent film of chromium oxide, whereas that of nickel is to produce an alloy in which only one crystal type (austenite) is present. As the chromium content is increased above 18 per cent progressively greater amounts of nickel are required to retain the austenite. When the chromium content is 20·5 per cent (as found in the analysed

screw) it is desirable to increase the nickel content to about 14 per cent (9·5 per cent in this screw).

Differential oxygenation—It must be remembered that an electric current can be set up between two parts of the same metal if one part is exposed to a greater concentration of oxygen in the surrounding electrolyte solution. The part that is more highly oxygenated will become cathodic, with consequent anodic dissolution or corrosion of the other piece. Differential oxygenation can occur in two ways *in vivo*. First, one screw may lie in scar tissue and another in granulation tissue, tissues which may differ in oxygen potential. Second, a crevice may be formed between two pieces of the same metal in contact with one another. It then becomes more difficult for oxygen to diffuse into the crevice than for it to reach the metal surface. The crevice thus becomes anodic, and tends to corrode.

MISCELLANEOUS FACTORS

Other factors which may well play a minor but contributory role in producing electrolysis exist. Small temperature changes between one end of the plate-screw system and the other can be shown *in vitro* to set up voltage changes between these points. Such temperature differences are possible in the body between subcutaneous plates and more deeply buried screws.

CORROSION IN PRACTICE

It is debatable whether " electrolytic inflammation " can interfere greatly with the healing of the internally fixed fracture. It is our experience that such " inflammation " usually occurs at some distance from the fracture site—for example, around one of the screws—and is

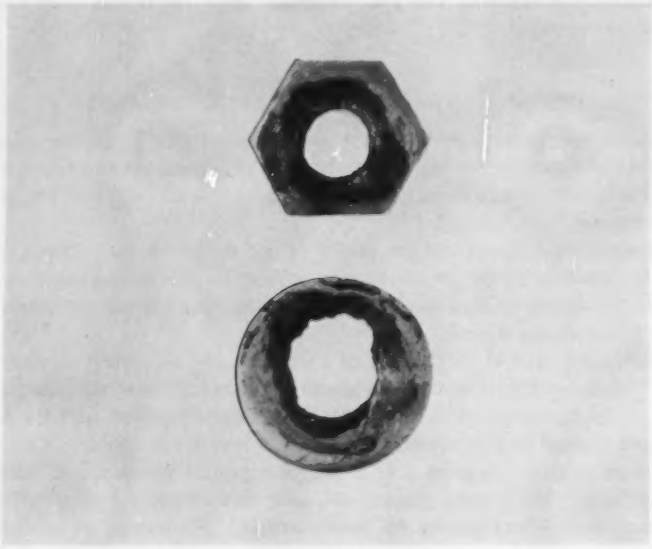

FIG. 12
Nut and washer showing marked corrosion of internal parts.

walled off by fibrous tissue. Here a " chemical abscess " is probably formed and, especially if the plate is subcutaneous, this abscess may point through the skin and become secondarily infected. We have not infrequently seen primary skin healing of the plated fracture without any evidence of sepsis, but with localised wound breakdown six or eight weeks later, which may be caused in this way. Such " chemical abscesses," even without secondary infection,

eventually lead to the loosening of the screw and loss of fixation. Yet not all screws loosen; indeed some defy attempts at removal. But all screws loosen if the fracture fails to unite. What part of this loosening is electrolytic and what part is due to mechanical stresses in any particular instance is impossible to assess, but it would appear that loose screws can lead to non-union and non-union to loose screws.

Chemical differences between individual screws and between screws and plates certainly exist. These differences become particularly important if it is the practice of the theatre sister to place plates and screws in a common box, for these plates and screws from different batches, or indeed from different manufacturers, may be selected for use in one particular case. They may differ considerably on chemical analysis, with resultant voltaic action.

Even when chemically identical materials are used they may not be in the same metallurgical condition. The threaded portion of a screw or nut may, by mechanical distortion, have been rendered anodic. When they are bolted together other factors will operate to concentrate the amount of corrosion at their points of contact. These factors are the destruction of the protective oxide film by the mechanical abrasion of one thread upon another, and the differential oxygenation effect which tends to make any crevice between them anodic. Corrosion at this point may rapidly destroy the rigidity of fixation. Indeed, an instance has been encountered in which the internally threaded part of a nut has been completely dissolved away (Fig. 12). It seems, therefore, that wherever possible, bolted structures should be avoided, as corrosion occurs at the very point where the mechanical strain is greatest.

A similar but less pronounced effect is obtained between the region of contact of the head and neck of the screw and the plate. Bony dissolution in this region readily causes loss of fixation. Electrolytic changes at this site are much more important than those occurring around the slot of the screw.

THE PREVENTION OF ELECTROLYTIC CORROSION

Clearly the most important points in the prevention of electrolytic corrosion are the use of identical metals in exactly the same metallurgical condition, and the careful handling of these metals both before and during use, to avoid abrasion of their surfaces and their mechanical distortion.

We insist on a rigid control of the purity of the drugs we use. This control must be extended to the chemical and mechanical standards in the manufacture of orthopaedic materials. (In the course of this study we have noted also marked mechanical differences in supposedly identical materials.)

Ideally, all metals used in the fixation of a fracture, and all metals coming into electrical contact (which implies mechanical contact) should be chemically and metallurgically identical. The clamps and the screwdrivers should be uniform in composition with the screws and the plates. This is a counsel of perfection, but perfection is a worthy aim.

There is little prospect at present of a completely inert substance of sufficient strength for use in the body. No known plastic will take the necessary strains without breaking, even if one uses a steel core within the plastic article. We have tried to reduce corrosion by coating the plates and screws with silicones, which provide an electrically insulating coating, but unhappily this easily chips off during the insertion of the screws, and thus fails to achieve its object.

It is important to remember that dissimilar metals will only act as a voltaic cell if they are in electrical contact at some point. It might be worth while considering the insertion of a thin insulating washer between the plates and the heads of the screws. We have at present no experience of this, but it is noteworthy that we have rarely observed corrosion in the isolated screws holding a bone graft.

SUMMARY AND CONCLUSIONS

1. The factors producing electrolytic corrosion of stainless steels are reviewed, and it is shown how several factors operating together can accentuate corrosion in certain positions in the plated fracture.

2. These factors can be minimised by good metallurgy and good engineering on the part of the manufacturers.

3. When and if materials become metallurgically satisfactory, or a truly inert substitute for metal is discovered, it will be possible to assess the true contribution of infection, faulty operative technique and mechanical factors to the failures of internal fixation. Such a critical assessment of these probably more important factors is at present bedevilled by uncertainty as to the purely physical condition of the materials as supplied to the surgeon.

We would like to thank Mr D. Ll. Griffiths, the Director of the University Department of Orthopaedic Surgery, in which this work has been carried out, for his interest and advice; also the Departments of Medical Illustration and Pathology for their photography.

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INTERNAL PROSTHESES

The Problem in Relation to Materials*

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During the last twenty years advances in various branches of medicine have made possible more extensive operative procedures involving the replacement or repair of tissues by prosthetic devices. An increasing number of apparently suitable materials but with widely differing properties have become available to the surgeon and appliance manufacturer. While there have been many apparent successes with implants, there have also been a number of failures. It is important not to reject out of hand the failures, because much valuable information can be gained from an analysis of these cases which may help in the future to prevent the misapplication of materials.

Prostheses may be required to be permanent or temporary, weight bearing or non-weight bearing, free or fixed. Whatever the purpose of the implant, the following interrelated factors must always be considered: 1) the properties of the material used; 2) the forces to which the implant will be subjected; 3) the response of the tissues of the patient in relation to the implant; 4) the surgical technique to be employed.

It is only possible here to look at some of the facets of what may be termed the " prosthetic problem."

NON-METALLIC IMPLANTS

If polymethyl methacrylate, Nylon, polythene, stainless steels or chrome-cobalt alloys in the form of pellets, five millimetres long and three millimetres in diameter, are implanted in the muscle of guinea pigs, there is a minimal tissue response. A thin, non-adherent, fibrous tissue envelope is formed which may be thickened in relation to any sharp edge. No inflammatory cells are found, there is no increased vascularity and no change in the surrounding muscle. The implants are apparently tolerated by the host tissues and are not in themselves modified by the action of tissue fluids. They can be said to be biologically suitable materials. However, they are not subjected to any significant mechanical forces. The resistance of plastics to abrasion is low compared with that of bone or metals, and if these polymers are used for weight-bearing hip prostheses, the tissue reaction to the implant may be very different. Abrasion of the prosthesis may occur, with the formation of considerable quantities of particulate material. The quantity and size of these particles depends on the polymer used, the activity of the patient, whether the acetabulum has been reamed, the accuracy of fit of the implant, and the method of sterilisation of the prosthesis. In the hip, abrasion of acrylic resins results in the formation of particles which are probably colloidal in size. It has never been possible to demonstrate with certainty powdered polymer in those cases in which there has been extensive wear of the prosthesis. Considerable fibrous tissue formation is, however, often found in association with worn prostheses.

With Nylon, which has a surface hardness approximately one half that of acrylic resin, most particles formed are of the order of 1 to 10μ in size although larger fragments of up to 300μ in size may be produced. The tissue reaction is different from that found with acrylic resins. At operation, on opening the joint capsule, which is distended by synovial fluid, collections of proliferating tissue are found in saucer-like depressions in adjacent bone (Fig. 1). Histological examination of the tissues shows that there is a proliferative histiocytic

* Paper read at a meeting of the British Orthopaedic Association in Liverpool, October 1955.

response, the particulate Nylon being phagocytosed by the histiocytes (Fig. 2). Zones of necrotic material, separated by vascular fibrous tissue, contain large concentrations of Nylon particles (Fig. 3). From the periphery of the zone towards its centre the histiocytes can be seen in various stages of dissolution.

With polythene, a material which has a surface hardness approximately one half that of Nylon, the particles produced by abrasion vary in size from 30μ to 1000μ. The capsule of the joint is distended with synovial fluid and there is erosion of bone. Histologically the foreign-body giant cell is conspicuous. Zones of necrosis in the reactive tissue are not found. The degree and type of cellular response is dependent on the abrasion-resistance of the implant.

FIG. 1
Destruction of head and neck of femur and roof of acetabulum, after insertion of a nylon hip cup one year before.

FERROUS ALLOYS

Problems still arise when metals, particularly the ferrous alloys, are used in place of plastics. All implants are liable to corrosion to a greater or lesser degree. The causes and mechanism of corrosion are complex and often not clearly understood. Iron rusts in the presence of oxygen; that is, a film of iron oxide forms on the outer surface, rapidly at first, but at a gradually decreasing rate as the oxide film shuts off the remainder of the iron from the oxygen. If now the metal is placed in water in the presence of carbon dioxide and other electrolytes, a further change becomes possible. The iron oxide film is destroyed and there is a passage of metal as cations into solution. Although oxygen may be present in solution it is not able to reform the oxide layer fast enough. The process of corrosion is accelerated when substances of differing electrode potential are present in the system. A familiar example is the dissolving of the zinc electrode in a voltaic cell. Some metals, for example chromium, form a stable oxide layer in air which protects them from further corrosion even when they are placed in an electrolyte, providing that this surface oxide layer is not damaged. By incorporating 18 per cent of chromium in ferrous alloys their resistance to corrosion is greatly enhanced. The addition of nickel and molybdenum, while still further contributing to their

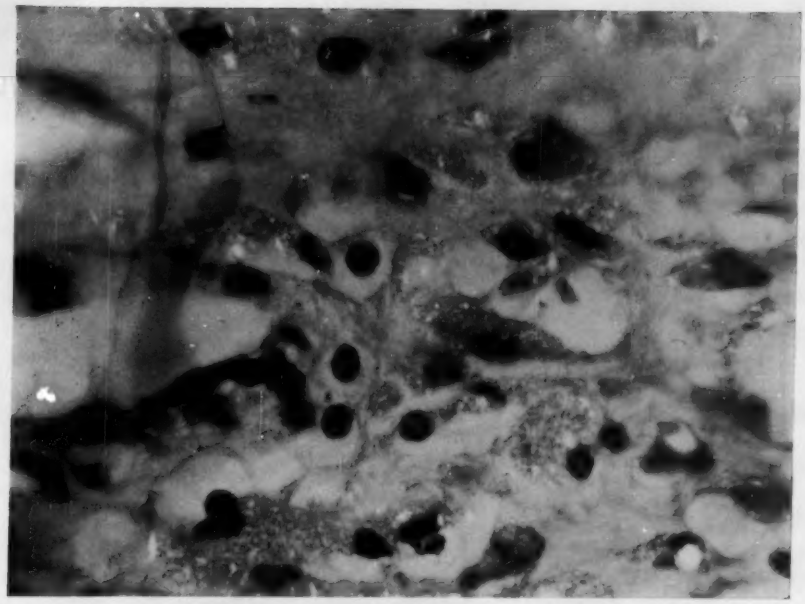

FIG. 2
Histiocytes loaded with birefringent particulate nylon. (M.300. ×430. Crossed polaroids.)

FIG. 3
Section of tissue from joint showing zones of necrosis separated by vascular fibrous tissue. Around the periphery of the necrotic area are found histiocytes containing particulate nylon. (M.302. ×12. Partially crossed polaroids.)

resistance to corrosion, improves their mechanical properties. Steels containing 18 per cent of chromium, 8 per cent of nickel and 2 per cent of molybdenum are known as 18-8-SMo in America and in this country as EN58J (Firth Vickers F.M.B.). The full effect, however, of these passivating agents, as they are termed, can be achieved only if care is taken in the handling and treatment of the alloy during all stages of manufacture of the implant, and at the time of implantation in the patient. The polishing and surface finishing of the implant, which may be carried out by electro-chemical or mechanical means, is all important. Many implants removed from patients showing macroscopic signs of corrosion are found to have a poor surface finish, small fissures and pits having been left which have become centres of corrosion. All alloys in their solid state are crystalline in nature. Crystal growth occurs while a metal is freezing into a solid. The crystals are known in metallurgy as grains, and when coalescence of the grains occurs, grain boundaries are formed. If the rate of growth and size of grain is not carefully controlled, considerable grain stress may result. The grain size is one of the factors determining the physical properties of an alloy. It is known that the boundaries of stressed grains are more liable to corrosion, one reason probably being that at the surface of the implant the chromium oxide layer is either strained or not continuous.

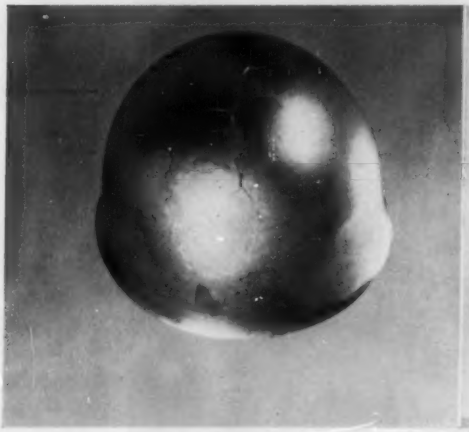

FIG. 4

FIG. 5

Failed Smith-Petersen cups. The cup shown in Figure 5 was inserted five and three-quarter years previously.

Stressing of grain boundaries can result from methods of cold working the alloy or by overheating during milling, drilling or grinding operations, by distortion of an implant to make it fit the patient, or by excessive stressing of an implant within the patient.

Fatigue—Scales and Zarek (1954), have pointed out that all implants are liable to fatigue—that is, to the action of alternating stressing to a greater or lesser degree. As a result the grain boundaries become stressed and corrosion may result. The term corrosion fatigue is used to describe the simultaneous action of corrosion and fatigue. Before a metal is inserted in the body, stress may be relieved by heat-treating or annealing and, if this process is carried out under carefully controlled conditions, molecular reorientation of the distorted structure of the crystal can occur. Over-annealing, however, will result in a change of grain size and a lowering of strength, hardness and ductility of the metal. Professor Mercer and Mr Wheble have very kindly sent me stainless steel hip cups that have failed (Figs. 4 and 5). It is difficult from the radiographs of Mr Wheble's patient to see how long after insertion the failure occurred, and in any case it is often impossible to detect cracks in hip cups radiographically (Fig. 6). Both the cups are of an austenitic variety of stainless steel. In both cases corrosion fatigue is probably the cause of the failure. The whole thickness of

the spherical part of the cup appears to be somewhat below that usually found. There is no evidence of gross wear and presumably the thinning occurred during manufacture.

The hip cup is particularly liable to be heavily stressed, as it is a mobile joint interposition which is both hemispherical and cylindrical. When downward tilting of the cup occurs, the skirt impinges on the calcar femorale, the cylindrical portion being brought into contact with the spherical sectioned acetabulum. No longer is the cup a floating member, but rather a fixed member which is subject to high localised alternating stressing both at the base of the skirt in contact with the calcar and in the region of the junction of the spherical portion of the cup with the cylindrical skirt.

Use of dissimilar metals—When dissimilar steel alloys are placed in the tissues, corrosion due to a difference of electrical potential with a transference of ions can occur (Venable and Stuck 1938). Changes also occur in the surrounding tissues which have been described

FIG. 6
Radiograph of the cup seen in Figure 5 showing only one crack passing through the skirt of the cup.

by Fink (1944) as "electrolyte inflammation." There is a local increased vascularity with rarefaction of bone; inflammatory cells and fibroblasts appear, and deposits of intra-cellular and extra-cellular iron can be demonstrated. In August 1954 three Moore's pins were inserted into a slipped upper femoral epiphysis of a boy aged twelve. Two months later an area of rarefaction could be seen where two of the pins were adjacent to each other. On the radiograph a notch on one of the pins indicated that corrosion was taking place. There was rarefaction of bone with inhibition of callus formation at the outer cortex of the femur around the nuts at the distal ends of the pins (Fig. 7). In January 1955 the pins were removed. It was found difficult to remove one of the pins because it broke at the notch, but after extensive chiselling the proximal part was withdrawn. The nuts were brightly polished and were non-magnetic and showed no macroscopic evidence of corrosion. The pins, however, were strongly magnetic and poorly polished, and two of them were badly corroded; the threads

at their proximal ends were dull. This case illustrates what may happen when dissimilar alloys are used, and this is not an isolated case.

Even when every care has been exercised in the design, manufacture and use of stainless steel implants, it is sometimes found that callus formation is inhibited, the implant becomes loose or breaks. Stainless steel screws are often preferred to those of Vitallium or Vinertia because it is said that they can be taken out more easily. This is true because stainless steel implants are usually surrounded by a layer of some thickness of fibrous tissue, the bone failing to form in such intimate contact with the structure as to key or lock it in position. Bowden, Williamson and Laing (1954), using an isotope technique, investigated the transference of metals from screwdrivers and spanners, and showed that appreciable quantities of metal were transferred to the implant. Spectroscopic examination of the surrounding tissues revealed that in those areas in contact with the instrument-handled part of the implant there was an abnormally high metallic concentration. Shaw and Scales (1954) investigated the transference of metal from radioactive mechanically operated tools used to cut and drill bone and found a transference of metals as shown in Table I.

These investigations suggest that some past failures with stainless steel implants may be explained on the basis of transfer of dissimilar metals from instruments, used at the time of operation, to implants. (For example, saw blades are made from Firth Vickers F.H. steel and drills from F.S.T., while others are produced from ferrous alloys of a non-stainless variety. Saw blades cannot be made from F.M.B. steel to wear nearly as well as those made from F.H. steel.)

FIG. 7

Radiograph showing areas of rarefaction of bone around nuts on Moore's pins. Area of rarefaction with corrosion of one pin can be seen where two of the pins approximate.

CHROME-COBALT ALLOYS

In view of these problems with the ferrous alloys, why is it that they have been used for so long, and what are the alternatives? Until recently, the manufacture of chrome-cobalt alloy implants was controlled by the Austenal Laboratories in America and a great deal of technical information has been amassed by them. These alloys are difficult to cut, drill or machine, and hence the implant has to be cast by the lost-wax technique. In this process a wax pattern, produced by injecting wax into a metal master mould, is invested in a refractory medium and the wax melted out leaving a cavity into which is cast the alloy. For the individual prosthesis a pattern in metal or plastic has to be made and an impression with either a solid or flexible medium taken. From this a number of individual wax positives can be prepared. With a casting technique, radiographic inspection of the casting is important to detect voids and cracks which often occur. This results in a number of rejects which adds to the cost of the implant.

Our own industry, while willing to help in this work, is given little encouragement to undertake what is relatively a costly process of manufacture, and thus the facilities for the production of the individual or experimental chrome-cobalt alloy prostheses do not exist in this country to the same extent as in the United States.

It will never be possible to assess the number of failures of implants, but I know of no

case of failure of a chrome-cobalt alloy from corrosion. A further advantage in the use of these alloys is that surgical instruments can be tipped with the same alloy, and thus the problems associated with the transfer of metals would probably cease to exist. As these alloys have much greater resistance to corrosion than the ferrous alloys, implants do not need to be polished, and unavoidable surface damage does not result in corrosion.

In the future, metallic elements such as Titanium and Zirconium may offer possibilities. It might seem preferable to use elements rather than alloys because one would only be dealing with problems of a single and not a compound material.

TABLE I

TRANSFER OF METAL FROM SURGICAL TOOLS TO BONE

Operation	Approximate weight of swarf (*milligrams*)	Weight of steel per 100 milligrams (μgrams)	Weight of steel adhering to bone (μgrams)	Weight of steel adhering after brushing (μgrams)
A	300	91	40	—
B	250	290	300	160
C	240	18	7·5	—
D	140	28	8·5	—

Specimens of bone taken from bone bank and allowed to thaw.

A Two transverse cuts through hemi-section mid-shaft of tibia, marrow removed, swarf pooled, one face counted.
B Three transverse cuts through mid-shaft of femur, marrow not removed.
C Two holes drilled transversely through cancellous part of tibia, swarf pooled, bone sectioned across both holes, one face counted.
D One hole through both cortices of fibula, bone sectioned through hole, one face counted.
Drill, $\frac{5}{64}$ inch diameter, of stainless steel, Type F.S.T.
Saw blade, 2 inches diameter, of stainless steel, Type F.H.

MECHANICAL AND TECHNICAL PROBLEMS

The satisfactory use of materials can only be established by a study of their mechanical properties, coupled with investigations into the biological response of the cell in contact with the implant. The chemico-physical properties of the implant are partly dependent on the effects produced by the forces acting on the implant. Thus in their design, the magnitude and direction of application of these forces, after a surgical procedure, must be taken into account. All bones are struts which are relatively weak, and that they are capable of transmitting forces of such a high value is because of balanced muscle control, design of joint at which movement occurs, and the stabilising effects of ligaments on these joints. Many implants are used for temporary fixation, and when bony union occurs they become purely passive members, subject to little stress. Other implants, such as a knee joint, are required to remain as active components for the life of the patient. Thus the method of attachment of the implant to the bone, the action of muscles and the reconstitution of their attachments to the implant, are aspects of the prosthetic problem which demand careful consideration. The possibilities of surgical technique have always to be borne in mind. An implant is of little value if it is found at operation that in order to insert it extensive resection and transposition of tissue is necessary. For example, it is mechanically undesirable to plate a fracture of the shaft of the femur with one plate and four or six screws. It is technically much more difficult, however, to use bilateral slotted plates and bolts which mechanically would be the more desirable method.

There is no doubt that unless the resources of other branches of science, particularly engineering, are brought to bear on many problems, there will be an ever increasing number

of implant failures. Standards of composition, design and manufacture of existing implants, in everyday use, do not exist in this country, although in the United States they are being developed. These standards are necessary to safeguard both the patient and the surgeon. New materials are continually being produced and methods of manufacture are changing. Thus standards will have to be revised after systematic experiment and clinical observation. Some day a start must be made on this problem. The longer it is deferred, the more complex it becomes.

I would like to acknowledge the receipt of a grant from the Medical Research Council who have supported certain aspects of the work connected with the problems discussed. I would also like to thank Mr H. T. Shirley of the Brown-Firth Research Laboratories who kindly carried out the spectrographic analyses of the metals.

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MEASUREMENT OF THE VARIATIONS IN CALCIFICATION IN NORMAL RABBIT BONE

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It is now well known that the distribution of mineral salts in bone tissue is not uniform. This is strikingly demonstrated in microradiographs of transverse sections of bone (Amprino and Engström 1952, Amprino 1952, Jowsey, Owen and Vaughan 1953). Quantitative measurements have also been made of this variation in content of mineral salts using the microradiographic-photometric technique developed by Engström. Previous workers (Amprino and Engström 1952, Amprino 1952) have been particularly interested in the relative amounts of mineral salts in Haversian systems and have determined the variation of calcification within the systems themselves and with respect to their more highly calcified surroundings. Measurements made on sections from bones of a large number of species showed that the amount of mineral salts in the Haversian systems is from 0 to 20 per cent lower than their surroundings and that the more lowly calcified systems predominate in younger animals. Also the calcification within the systems themselves is not uniform and depends on the age of the system.

A detailed investigation has already been made of the growth and structure of the proximal half of the rabbit tibia from six weeks to seven months old using both microradiographic and autoradiographic techniques (Owen, Jowsey and Vaughan 1955). Microradiographs of serial transverse sections from the mid-shaft to just below the epiphysial plate of the tibia showed clearly, though qualitatively, that different structures in the bone have varying degrees of calcification. More quantitative measurements of these differences have been made, and will be described.

It was previously shown (Owen, Jowsey and Vaughan 1955) that the remains of epiphysial bone, which was presumably formed when the epiphysial plate was at a particular level, was to be found at practically all levels at all ages studied. Resorption cavities and developing Haversian systems at all stages of calcification (the latter having been formed within resorption cavities) were situated almost entirely within this epiphysial bone, indicating its gradual removal and replacement as part of the remodelling process. The epiphysial bone was of lower calcification than the bone later formed on its periosteal and endosteal surfaces and could easily be distinguished, especially in the older animals where there had been considerable deposition of new bone on these surfaces. (Endosteal bone actually being laid down at the time the animal was killed appeared as a narrow band of lowly calcified bone external to the older highly calcified endosteal bone.) Remnants of calcified cartilage were embedded in the epiphysial bone and these showed a much higher absorption of x-rays than either the bone adjacent to them or the more recent periosteal and endosteal bone. These features are shown, for example, in Figure 1, which is a microradiograph of part of the internal wall of a section from the mid-shaft of a seven months old rabbit. In the present investigation measurements have been made of the relative content of mineral salts in calcified cartilage, bone immediately adjacent to the latter, and periosteal and endosteal bone, in rabbits which were six weeks, three and a half months, and seven months old.

EXPERIMENTAL METHOD

The experimental technique used is similar to that described by Engström and Welin (1949) and is briefly as follows. A microradiograph of both a transverse section of bone and

a step-wedge of foils of known composition and thickness was obtained. By comparing photographic densities the absorption of a particular structure in the bone could then be obtained in terms of the thickness of the foil. Since the sensitivity of the photographic emulsion depends on the wavelength of the x-rays, the step-wedge must be of a composition such that the same fraction of x-rays transmitted by various thicknesses of bone and foil has the same wavelength distribution over the range used. Using the composition of bone as given by Engström (1946) it was calculated that this condition was satisfied to within 1 per cent by aluminium foils for the present wavelength range—that is, the mass absorption coefficient of bone was for all practical purposes proportional to the mass absorption coefficient of aluminium over the wavelength range used.

To obtain microradiographs with good contrast between regions of bone containing different amounts of the calcium salt, it is preferable to use x-rays of wavelength just less than 3·06 Å, which is the position of the K-absorption edge for calcium. This is because,

FIG. 1

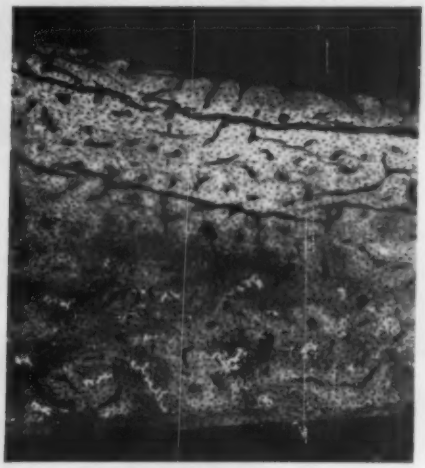

FIG. 2

Figure 1—Microradiograph of part of section of internal wall from midshaft of seven months old rabbit tibia. Note central band of epiphysial bone containing *a*) remnants of highly calcified cartilage, *b*) " bone adjacent to cartilage " showing low degree of calcification, *c*) several completed osteones (*e.g.*, A) of higher degree of calcification. Note bands of highly calcified bone on periosteal (*lower*) and endosteal (*upper*) edges. (×50.) Figure 2—Microradiograph of part of section of anterior wall from metaphysis of seven months old rabbit tibia. Note band of epiphysial bone on periosteal (*lower*) edge containing *a*) remnants of highly calcified cartilage, *b*) " bone adjacent to cartilage " showing low degree of calcification, *c*) completed osteones of higher degree of calcification. Note band of highly calcified bone on endosteal (*upper*) edge. (×50.)

for these x-rays, the absorption coefficient for calcium is high compared with that for other constituents of bone. The same x-ray tube was used as in our previous work (Jowsey, Owen and Vaughan 1953). The microradiographs for the present measurements were obtained with 20 kev radiation from a chrome target filtered through a 25 micron aluminium window. A graph of this radiation in roentgens as a function of wavelength is shown by curve A in Figure 5. The amount of the radiation transmitted by " bone " (Engström 1946) and by aluminium was calculated; and, for the range of bone sections used (40 to 60 microns), 1) there was a linear relation between the thicknesses of bone and aluminium which transmitted the same fraction (to within 1 per cent) of the x-rays; and 2) the wavelength distribution for the same fraction of transmitted radiation agreed closely for the two substances. A typical illustration of this is shown by curves B and C in Figure 5, which show the same fraction of x-rays transmitted by 50 microns of bone and 33 microns of aluminium respectively. For

wavelengths less than 3·06 Å the agreement was practically identical; for wavelengths greater than 3·06 Å a small portion of x-rays is transmitted by bone and not by aluminium. This was neglected, however, since it was always less than 1 per cent of the total. The absorption of x-rays by various structures in the bone can therefore be compared directly in terms of the thicknesses of aluminium foil which absorb the same amount of x-rays. For the above radiation it can also be shown that the inorganic part of bone, which is commonly assumed

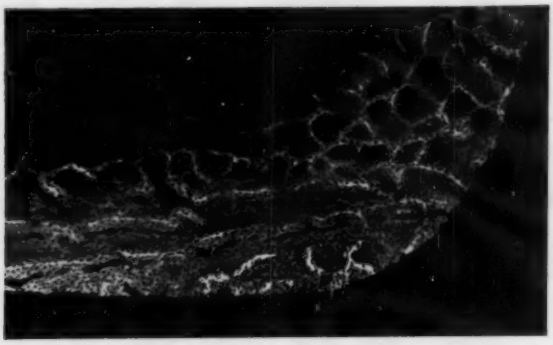

FIG. 3

Microradiograph of corner between internal and posterior walls from junction of metaphysis and shaft of six weeks old rabbit tibia. Remains of epiphysial bone containing highly calcified cartilage on periosteal side of corner. Active endosteal deposition; note highly calcified network enclosing Haversian systems of low calcification. (×50.)

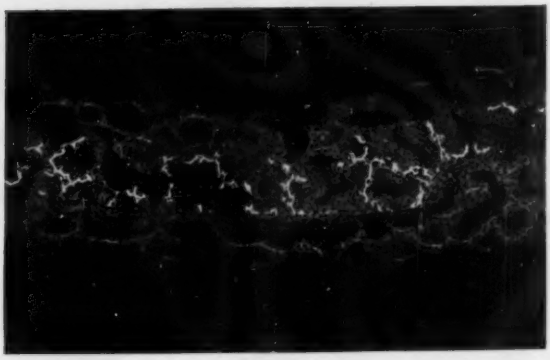

FIG. 4

Microradiograph of part of anterior wall from junction of metaphysis and shaft of six weeks old rabbit tibia. Mainly epiphysial bone containing remnants of highly calcified cartilage. Some periosteal and endosteal deposition. Note highly calcified network enclosing Haversian systems of low calcification. (×50.)

to be a hydroxy-apatite, accounted for about 90 per cent of the total amount of radiation absorbed in roentgens, the other 10 per cent being absorbed by the organic matrix. Differences in the absorption of the x-rays are therefore due mainly to differences in the content of the mineral salt.

The rabbit tibiae were embedded in perspex. Sections about 100 microns thick were cut on a milling machine and then ground by hand under alcohol on ground glass to a thickness of about 50 microns. The section was placed alongside the step-wedge of aluminium

foils (20, 30, 40, 60 and 80 microns thick) on a Kodak maximum resolution plate and pressed in contact with it. A sheet of Styrafoil (12 microns thick and for our purposes transparent to x-rays) was placed over the top and screwed down with a brass ring to keep the section and step-wedge in position. It is necessary that the portion of the x-ray field used should be uniform, and in order that a large enough area should be available the plate was placed about 13 centimetres from the target. At this distance an area of approximately 0·5 by 0·7 centimetre was of uniform intensity to less than 1 per cent. The space between the x-ray window and the plate was evacuated. With a tube current of 220 microamps the exposure time was of the order of ten minutes. With these conditions the photographic densities on the microradiograph of the bone were in the range 1·0 to 1·8. After exposure the plates were developed for three

TABLE I

MEASUREMENT OF RELATIVE CALCIFICATION EXPRESSED AS THE RATIO OF X-RAY ABSORPTION IN CALCIFIED CARTILAGE TO "BONE ADJACENT TO CARTILAGE," PERIOSTEAL BONE AND ENDOSTEAL BONE, IN DIFFERENT PARTS OF THE TIBIA FROM RABBITS OF DIFFERENT AGES

Age of rabbit	Position of section	Ratio of x-ray absorption in	
7 months	Mid-shaft	Calcified cartilage / "Bone adjacent to cartilage"	1·19±0·03
7 months	Mid-shaft	Calcified cartilage / Periosteal bone	1·08±0·03
7 months	Metaphysis	Calcified cartilage / "Bone adjacent to cartilage"	1·18±0·03
7 months	Metaphysis	Calcified cartilage / Endosteal bone	1·07±0·03
3½ months	Metaphysis	Calcified cartilage / "Bone adjacent to cartilage"	1·18±0·04
3½ months	Metaphysis	Calcified cartilage / Endosteal bone	1·10±0·03
6 weeks	Mid-shaft	Calcified cartilage / Periosteal bone	1·11±0·04
6 weeks	Metaphysis	Calcified cartilage / "Bone adjacent to cartilage"	1·24±0·05
6 weeks	Metaphysis	Calcified cartilage / Endosteal bone	1·09±0·04

and a half minutes in Kodak D178 developer at a constant temperature of 20 degrees centigrade, washed and immersed in an acetic acid stop-bath for thirty seconds, fixed for fifteen minutes and finally washed for several hours.

Great care was taken in choosing the part of the bone section on which measurements were to be made. Preliminary microradiographs indicated the parts of the section which were most uniform in thickness and free from cracks or other artefacts. The thickness of the section was measured with a Baty comparator which had a measuring foot with a cross-section of diameter of 1 millimetre and which could be read to one micron. Only those parts of the section were chosen for measurement whose surroundings gave the same thickness reading to within a distance of 1 millimetre. Different structures in the bone section whose calcium salt content was being compared were never more than 300 microns apart, and it is unlikely that the thickness of the section varied over this distance. For a large number of measurements the comparator gave readings which were within ±2 per cent.

The areas whose densities were to be measured on the microradiograph were of the order of 30 microns in diameter, hence a densitometer for determining the density of areas just less than 10 microns in diameter was constructed. A diagram of the instrument is shown in Figure 6. L is a light source giving a constant output, C is a condenser lens, the specimen S is a microradiograph, a magnified image of which is formed on the white screen D by the lens O. The area on the microradiograph whose density is being measured is defined by a pinhole, half a millimetre in diameter, in the centre of the screen D. Light passes through the pinhole and is detected by the photomultiplier P giving a current reading on the galvanometer G. The area on the microradiograph corresponding to that covered by the pinhole is just less

TABLE II

MEASUREMENT OF CALCIFICATION EXPRESSED IN TERMS OF THE NUMBER OF MICRONS OF ALUMINIUM FOIL WHICH ABSORB THE SAME AMOUNT OF X-RAYS AS A 50μ THICK SECTION OF A PARTICULAR STRUCTURE OF BONE

Age of rabbit	Position of section	Number of sections measured	Total number of measurements	Bone structure	Microns of aluminium equivalent to 50 microns of bone
7 months	Mid-shaft	3	12	Calcified cartilage	48·9±1·9
7 months	Mid-shaft	3	15	" Bone adjacent to cartilage "	41·5±1·8
7 months	Mid-shaft	3	33	Periosteal bone	45·4±1·7
7 months	Metaphysis	3	21	Calcified cartilage	48·4±1·9
7 months	Metaphysis	3	35	" Bone adjacent to cartilage "	40·6±2·0
7 months	Metaphysis	3	36	Endosteal bone	45·2±1·7
3½ months	Mid-shaft	1	10	Periosteal bone	43·3±2·0
3½ months	Metaphysis	2	9	Calcified cartilage	49·0±2·0
3½ months	Metaphysis	2	11	" Bone adjacent to cartilage "	41·6±1·8
3½ months	Metaphysis	2	10	Endosteal bone	44·4±2·0
6 weeks	Mid-shaft	2	10	Calcified cartilage	47·3±2·0
6 weeks	Mid-shaft	2	11	Periosteal bone	42·5±2·1
6 weeks	Metaphysis	3	12	Calcified cartilage	48·0±1·7
6 weeks	Metaphysis	2	10	" Bone adjacent to cartilage "	39·0±2·0
6 weeks	Metaphysis	2	12	Endosteal bone	44·3±2·0

than 10 microns in diameter and it can be seen in the mirror M via the viewing tube V. The density of a structure on the microradiograph was determined by comparison with the calibrated annular density wedge A which has a continuous variation from 0 to 3 over 360 degrees. Its position could be read on the graduated disc Q.

When measuring the photographic density corresponding to a particular structure of bone many readings were taken and averaged. This was necessary because there was a spread in the density measurements of up to 3 per cent due to the fact that even bone within the same structure is not homogeneous. For each plate a graph of D (photographic density) versus thickness of aluminium foil in microns was plotted. A typical example is shown in Figure 7. From this the absorption of a particular bone structure could be read off in terms of microns of aluminium foil. The error in measuring the photographic density was ±1 per

cent. With the present microradiographs it was therefore estimated that a variation of $\pm 1{\cdot}5$ per cent in the absorption of x-rays by bone could be detected.

RESULTS

The measurements made of the relative content of mineral salt in different parts of the bone have been expressed in the present study in two different ways. 1) As a ratio of the absorption of x-rays by the highly calcified cartilage to the absorption by other parts of bone—namely *a*) " bone adjacent to cartilage " (that is, epiphysial bone laid down on the highly calcified cartilage); *b*) periosteal bone; *c*) endosteal bone (Table I; Figs. 1 to 4) (that is, bone laid down on these surfaces around small vessels or marrow spaces). Since these ratios are derived from measurements made on the same plate they do not depend on measurements of

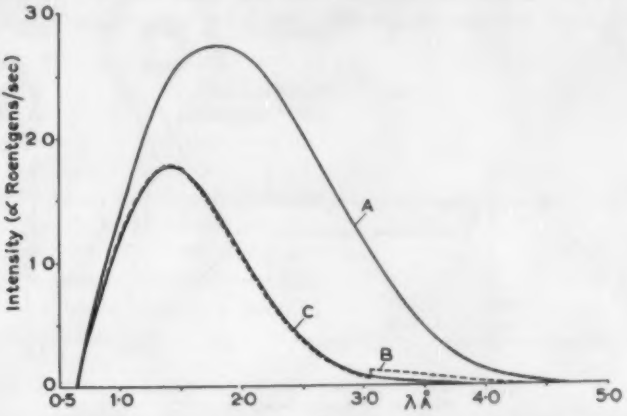

FIG. 5

Curve A shows the amount of 20 kev radiation in roentgens (arbitrary units) from a chrome target filtered by 25 microns aluminium. Curves B and C are for the same radiation transmitted through 50 microns of " bone " and 33 microns of aluminium respectively.

the section thickness. 2) In terms of the number of microns of aluminium foil that absorb the same amount of x-rays as a 50 micron thick section of a particular structure of bone (Table II). Assuming that rabbit bone has the same composition as that given by Engström, the average density of the bone structure can also be calculated from the present measurements, and for periosteal and endosteal bone in Table II this was 2·08 grammes per cubic centimetre. The errors in Table II include the 2 per cent error in measurement of the section thickness. The results given in the Tables are for the absorption of x-rays by both the inorganic and organic components of bone. A recent paper (Davies and Engström 1954) has shown that the amount of organic matrix is the same in both " young " and " old " Haversian systems even though they have different degrees of calcification. Assuming that the different structures observed here contain the same amount of organic matrix, then any difference in the absorption of x-rays is entirely due to differences in content of the inorganic salt. When allowance is made for the fact that a small fraction of x-rays is absorbed by the organic matrix the ratios in Table I are found to be increased by between 1–3 per cent. This is insignificant for the purposes of the present discussion and does not alter the values in Table II enough to affect the comparison between them that is given below.

As can be seen from Table I the ratio of x-ray absorption by calcified cartilage to that by " bone adjacent to cartilage " and to periosteal and endosteal bone are the same within

the experimental errors for rabbits of the three age groups. The absorption of x-rays by remnants of calcified cartilage found in both the mid-shaft and the metaphysis is the same within the errors for all three ages (Table II). " Bone adjacent to cartilage " was also calcified to about the same extent at all levels and all ages (Table II). It was noted that periosteal and endosteal bone is not evenly calcified but is more highly calcified in the spaces between the small Haversian systems (Figs. 1 to 4). All the values quoted in Tables I and II were from measurements made on these more highly calcified interstitial areas and within the errors are the same at all levels in all rabbits. In the older rabbits, three and a half months and seven months, the degree of calcification of the small Haversian systems in periosteal and endosteal bone varied only slightly from that of the spaces between them (Figs. 1 and 2). However, in the six weeks old animal where deposition of bone on the periosteal and endosteal surfaces is very active (Figs. 3 and 4), the bone surrounding the vessels or small marrow spaces is about 10–15 per cent less calcified than the bone laid down first, i.e., the network of bone in the interstitial areas between the systems. The latter have, within the errors, the same

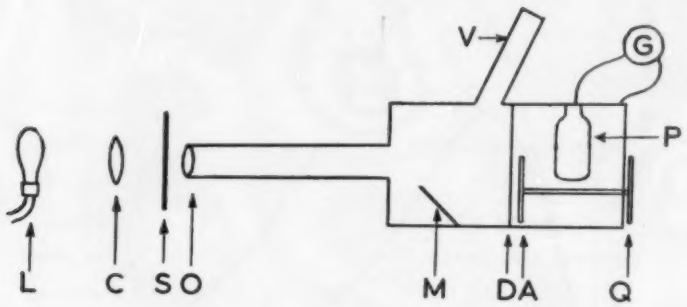

FIG. 6
Diagram of densitometer (see text).

calcification as bone in a similar position in the older animal. From autoradiographic studies of the uptake of Sr^{90} it is evident that, in the young animal, bone is being deposited very rapidly around these small vascular spaces on the periosteal and endosteal surfaces (Owen, Jowsey and Vaughan 1955) and it is likely that they quickly become highly calcified.

The major portion of seven months old bone consists of the periosteal bone deposited in the shaft and the endosteal bone deposited in the metaphysis (Owen, Jowsey and Vaughan 1955). At seven months the Haversian canals are on the whole smaller than in the very young animal and the bone has a more solid appearance and a fairly even degree of calcification. In bone from rabbits of this age, particularly, there was evidence that the more lowly calcified epiphysial bone had in some parts been replaced by completed osteones of the same degree of calcification as the neighbouring more highly calcified periosteal and endosteal bone, for example osteone A in Figure 1. These completed osteones are presumably Haversian systems which have been formed within a resorption cavity and which have gradually attained a high degree of calcification. Probably most of the epiphysial bone is gradually removed and replaced in this way during the life of the animal (Jowsey, Owen, Tutt and Vaughan 1955).

Two general conclusions may be drawn from the measurements recorded. 1) The bone of both young and adult rabbit has a framework of approximately the same strength or degree of calcification—that is, the supporting calcified cartilage formed below the epiphysial plate and the bone in the spaces between the small Haversian systems or vessels of periosteal and endosteal bone have the same degree of calcification in both young and adult rabbits.

2) There appears to be a trend towards uniform calcification throughout adult bone which is of the order of calcification of the interstitial areas of periosteal and endosteal bone. Evidence for this is the removal of lowly calcified epiphysial bone and its replacement by more highly calcified osteones.

SUMMARY

1. Measurements have been made of the relative calcification of different types of bone in tibia of the rabbit at the ages of six weeks, three and a half months and seven months by comparing their absorption of x-rays.

2. Calcified cartilage is between 8 and 10 per cent more highly calcified than periosteal and endosteal bone and about 20 per cent more highly calcified than bone formed immediately adjacent to cartilage.

3. Young and adult bones have a framework of approximately the same strength; that is, calcified cartilage, bone adjacent to cartilage and the interstitial areas of periosteal and endosteal bone have each approximately the same degree of calcification at all ages.

4. Adult rabbit bone approaches uniform calcification throughout, equal to the calcification of the interstitial areas of periosteal and endosteal bone. Evidence for this is the replacement of the lowly calcified epiphysial bone by osteones of higher calcification.

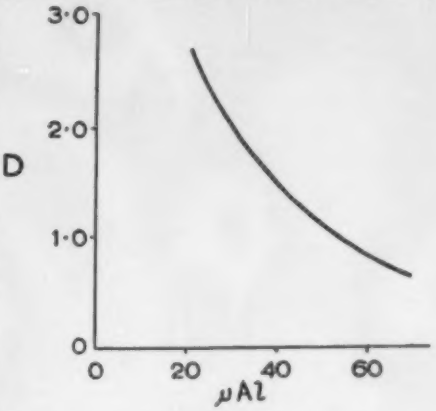

FIG. 7

Graph of density on photographic plate due to 20 kev x-rays transmitted by various thicknesses of aluminium in microns.

I am indebted to Dr D. G. Wyatt and Dr Janet Vaughan for advice and criticism.

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PROCEEDINGS AND REPORTS OF UNIVERSITIES COLLEGES, COUNCILS AND ASSOCIATIONS

GREAT BRITAIN AND IRELAND

BRITISH ORTHOPAEDIC ASSOCIATION

SPRING MEETING, 1956

The spring meeting of the British Orthopaedic Association was held at Gleneagles on May 3–5, 1956, with the president, Mr Philip Wiles, in the chair. The first day was occupied by the Instructional Course, the speakers being *Mr Roland Barnes* (Glasgow) on " Causalgia," *Mr P. H. Newmat* (London) on " Spondylolisthesis," *Mr L. W. Plewes* (Luton) on " Injuries in Industry," and *Mr Donal Brooks* (London) on " Injuries of the Brachial Plexus."

The morning of May 4 was devoted to short papers.

The effect of chemotherapy on the conservative treatment of spinal tuberculosis—*Mr J. P. Jackson* (Harlow Wood) said that of his eighty-nine patients 42·7 per cent had a tuberculous focus elsewhere, a number less than in other reported series where chemotherapy had not been used. They had been reviewed not less than two years after treatment, which consisted of immobilisation on a frame or plaster bed, streptomycin 1 gramme and P.A.S. 18 grammes daily for ninety days. Three patients died, all from meningeal or genito-urinary infection. Amyloid disease and sepsis were no longer problems, and sinuses closed more readily, although five had persisted and one new sinus developed under treatment. Posterior fusion by tibial grafts was usually performed when the lesion was quiescent, and in 36 per cent of these anterior fusion subsequently occurred spontaneously. Spontaneous fusion also took place in nearly half the patients not operated on, but this gave no certain guard against relapse, which occurred in 11 per cent of such cases. The relapse rate after surgery was only half that where no operative fusion had been performed. Abscesses were not opened as a routine, but it was found that only 24 per cent of those with persistent abscesses proceeded to bony fusion. He concluded that general treatment was still necessary, that conservative treatment with three months' chemotherapy gave 21 per cent unsatisfactory results, but that the total duration of treatment was shorter and mortality reduced. He considered that posterior grafting was well worth while. *Mr Robert Roaf* (Liverpool) pointed out the difficulties of comparing series treated at different hospitals. He remarked that chemotherapy was now given for nine to twelve months usually, and that we had learnt how fallacious the interpretation of radiographs could be in assessing quiescence.

V-nail fixation for trochanteric and basal fractures of the neck of the femur—*Mr A. W. Fowler* (Bridgend) recommended this operation, which was a modification of the " low nail " operation described by Brittain in 1942. After reviewing the objections to a nail-plate and to the long Smith-Petersen nail, he said that a V-nail (actually a clover-leaf Küntscher nail with the lateral leaves shaved down) provided stronger and safer fixation based on three solid points—the dense bone of the head, the upper end of the medial cortex of the shaft, and the outer cortex at the point of penetration. The only difficulty lay in passing the nail so obliquely through the outer femoral cortex, and for this he used a special cannulated drill that passed over the guide wire, and the nail was then driven along the track left by the drill. He preferred to use a cross pin to improve fixation of the lower end of the nail; provided the nail was not too long some collapse of the neck would not drive the point through the head even if extrusion of the nail was prevented. He permitted weight bearing in eight weeks, or earlier in stable fractures. He recounted certain technical difficulties and failures of this admittedly experimental method, but concluded that it was mechanically sound, cheaper and more efficient than pin or nail-plate, and one permitting telescoping to occur when necessary, but not coxa vara. *Mr G. K. McKee* (Norwich) had had similar favourable experience of the procedure, but remarked that some seemingly trochanteric fractures were really subtrochanteric posteriorly, so that a nail might split the cortex in this region. He therefore preferred to add to a vertical nail a slotted plate which would allow some telescoping. *Mr J. N. Wilson* (Stanmore) said that potential trouble from the cortical splitting mentioned by Mr McKee was prevented by the cannulated drill. The method was not helpful in subcapital fractures. He also liked a cross pin to help to stabilise the lower end of the nail in the necessarily large hole made for its introduction. *Mr Roland Barnes* (Glasgow) did not think the method was adequate for the unstable type of fracture because the comminution of the inner cortex would preclude stability and permit varus deformity with danger of the nail breaking through the head as in other methods.

Internal fixation in the treatment of Bennett's fractures—*Mr F. C. Badger* (Birmingham) said that it was difficult to maintain reduction by plaster splintage with embodied traction, and some redisplacement nearly always occurred. This had led some surgeons to treat them forthwith by mobilisation, but results were not fully satisfactory and later degenerative arthritis was certain. Simple suture of the rent in the dorsal capsule, as recommended by Gissane in 1953, was also unsatisfactory because tension led to stretching of the capsule and partial recurrence of deformity in ten of eleven cases. Internal fixation had been used but it had never become popular. The speaker, after discussing the mechanics of the injury and the causes of recurrence of deformity, described his method whereby the fragment was reduced through a dorsal incision and fixed by a small screw passed through the base of the shaft. No plaster was necessary. The patients were off work for an average of three weeks. Thirteen of seventeen patients had good results, the failures resulting either from the use of too short a screw with later redisplacement of the fragment, or from a badly placed screw. *Mr O. J. Vaughan-Jackson* (London) said that he used a small pin and a mid-lateral approach, and remarked that twice he had found the small fragment turned over so that the articular cartilage was apposed to cancellous bone on the major fragment.

Congenital dislocation of the hip—*Mr J. C. Scott* (Oxford) said that there had been little real advance in the last twenty-five years and there was wide variation in the percentage of good results claimed for present methods. He was reporting the interim results of seventy-five patients treated at Oxford since 1949 under the regime devised by Mr E. W. Somerville and himself. They were treated within what he called the "critical period"—from the beginning of weight bearing to the age of four. The method consisted in 1) gradual reduction by traction on a special frame, 2) operative removal of the limbus if this was shown by an arthrogram to be in-turned, and 3) a rotation osteotomy to correct anteversion when required. He believed that the dislocation was first anterior and later became superior and then posterior. He thought that it was usually the lesser trochanter that pushed in the limbus; the secondary reduction in circulation was responsible for the smallness of the epiphysial head and the later osteochondritis. The use of a frame was an essential part of the regime; the head of the femur could always be brought down but was often left standing out from the acetabulum. Seventy-five operations had been performed and only once had they failed to find an inverted limbus, which might be very large. The limbus could not be everted. The position of immobilisation depended on the degree of medial rotation required to reduce the head. Three weeks later a rotation osteotomy was done, the position of the upper fragment being unchanged and the shaft rotated to the neutral position. The plaster was removed after five weeks and early weight bearing permitted. The total duration of treatment had now been reduced to fourteen weeks. Three hips had redislocated either because the limbus had not been excised or because derotation had been insufficient and further operation was performed. All the hips had a range of movement 90 per cent or more of normal. He concluded by stressing that this was no place for light-hearted surgery and showed a film of the operations with radiographs of the first fifty cases.

Surgical treatment of slipping of upper femoral epiphysis—*Mr P. H. Newman* (London) reported the results in thirty-nine patients, of which twenty-three were excellent, eight good, none fair and eight poor. If there was less than one-third diameter posterior displacement and less than one-quarter diameter medial displacement the femoral head was pinned in situ. Because of the difficulty of inserting a Smith-Petersen nail into the epiphysis, the danger of damaging its blood supply and the possibility of a subtrochanteric fracture occurring at the point of insertion, he used instead a modification of Moore's pins. In this first group only one patient out of nineteen had a poor result. When the position was unacceptable an attempt was made to improve it by gentle manipulation and pins were inserted if adequate reposition was obtained. There were seven cases in this group, and although perfect reduction was seldom obtained the result was good in six. The single poor result was in a patient who had a stiff hip before the operation. When closed reduction failed open reduction through an anterior approach, with excision of a wedge of bone when necessary, was performed. A pin inserted in the neck before the open correction was begun could be driven into the head immediately it was reduced, thus preventing the possibility of loss of position during the later stages of the operation. Three of nine cases had poor results. Some other irreducible cases had been treated by subtrochanteric osteotomy, which he had now abandoned. He stressed that treatment in these cases was urgent. Poor results followed avascular necrosis and operations performed on hips already stiff from delayed diagnosis or prolonged traction. On these occasions two months' physiotherapy to mobilise the hip and improve the local blood supply was indicated before operation could be considered. *Mr A. M. Rennie* (Aberdeen) reported his results in fifteen patients. Three had developed avascular necrosis, and he agreed with Mr Newman that operations on stiff hips, which had resulted in two poor results in his own series, must be avoided. *Mr J. P. Jackson* (Harlow Wood) supported the speaker on the unsuitability of the Smith-Petersen nail for this operation; in three instances it had proved impossible

to insert it into the epiphysis. *Dr E. Thomasen* (Aarhus) said there were two groups, those of slow onset where the prognosis was better, and the acute group where there was greater danger of avascular necrosis. He had tried to stimulate fusion by drilling across the epiphysial plate, keeping the patient eight weeks in bed on traction without plaster fixation. Occasionally he had left a Kirschner wire across the plate for a few months as additional fixation. Subcapital wedge osteotomy with drilling and fixation by Kirschner wires was reserved for later cases: three out of fourteen had developed avascular necrosis.

Treatment of fractures of the neck of the femur by compression—*Mr John Charnley* (Manchester) said that with present methods one-third of femoral neck fractures failed to unite within the first year. He had been using a coarse-threaded screw in the femoral head; the smooth shaft of this screw passed through a socket inserted into the femoral neck and fixed by a plate to the femoral shaft. A nut and spring allowed compression to 25 pounds. The angle was fixed at 120 degrees, special tools were required, and while all other movements were prevented the spring maintained compression between head and neck should absorption of the latter occur. The apparatus permitted radiological measurement of any shortening taking place in the neck; and it was found that cases fell into two groups; in one shortening ceased after three months, while in the other it progressed for a year or more. In thirty-three cases available for study 82 per cent united satisfactorily. He thought the failure rate could be further reduced by technical improvements. Two post-mortem specimens showed that bony union and revascularisation of the head were greatest at the site of greatest compression. Radiographs were unreliable in the diagnosis of ischaemia in the early stages. He thought there were potential advantages in the mushrooming effect as the neck was pulled into the head, which, if dead, acted almost as a cartilage cap arthroplasty while the neck was driving through the necrotic head towards potentially living bone in the region of the ligamentum teres. He believed that in at least 66 per cent of fractures there was some interference with the capital blood supply, that his method eliminated non-union where the head was alive, and that its better mechanics reduced the ill-effects of ischaemia.

Arthrodesis of the hip by a lag screw—*Mr G. K. McKee* (Norwich) reported the results in fifty patients, all reviewed at least two years after operation. The operation was done through a lateral approach. After removal of the articular cartilage the hip was fixed by a lag screw inserted up the femoral neck and into the ilium. The neck was drilled out to allow the smooth shaft of the screw free passage when final compression was applied, and it was important to ensure that all the threaded part was in the ilium. The greater trochanter was removed and used as an ilio-femoral graft. In 76 per cent of cases no additional plaster fixation was used. The screw broke in eight cases, but in only half of these was it removed: this complication had not occurred in the last thirty-eight cases. There had been two deaths, one from delayed shock, the other from coronary thrombosis. Fusion failed to occur in only one case; this failure was ascribed to old ischaemic necrosis of the femoral head. Final results assessed on strict criteria showed 34 per cent excellent, 32 per cent good, and 28 per cent fair.

On the afternoon of May 4 an interesting and stimulating clinical meeting arranged by Mr I. S. Smillie was held at the Orthopaedic Unit, Bridge of Earn Hospital.

On May 5 the vice-president, Mr Andrew Fowler, took the chair.

Knock-knee in children—*Mrs M. Morley* (London), introduced by Mr H. J. Seddon, reported an investigation in which a thousand unselected children had been examined. They were divided into two groups, the first aged one to four years, the second five to eleven years; special reference was made to feeding, the provision of additional vitamins, weight, height, past illnesses, the degree of knock-knee, and the presence of ligamentous laxity, valgus ankles and flat feet. In general it was found that knock-knee, as reckoned by the intermalleolar gap when lying with the knees touching, became increasingly common and severe up to the age of three and a half years and then diminished steadily. Sex made no difference to the incidence, but the more marked the knock-knee the greater was the weight of the children. No correlation with height was found, and valgus " ankles " and flat feet (the latter almost universal in the very young) diminished with age and were unrelated to knock-knee. Mrs Morley had failed to find any association between knock-knee and the age of first walking, the duration of breast-feeding, additional vitamins, or the length of time spent in bed because of illness. She said she was not in a position to answer the question of *Mr G. Fisk* (Epping) as to whether the reduction of knock-knee with increasing age in girls was due to the widening of the pelvis, even though the angle between tibia and femur might remain constant.

Healing of experimental wounds of the flexor profundus tendon in the dog—*Mr M. S. Brett* (Stanmore) had undertaken this experimental work because results of repair in the digital sheath in man were so unpredictable. He had made an incision half way through the tendon at a site so chosen that by subsequent flexion and splintage in the selected position the wound of the tendon could be kept in

apposition to intact tendon sheath or to intact but avascular sublimis tendon, or alternatively be allowed to move freely unsplinted. Tendons were re-examined one, two or three weeks later. When splinted in contact with the vascular sheath adhesion occurred, mesothelial cells and later fibroblasts grew into the fine gap at the wound and vascularisation was marked. Almost no cellular reaction occurred in the tendon cells proper. In contact with the sublimis tendon mesothelial cellular reaction was less and again no reaction in collagen occurred within the three weeks. When the tendon was left free to move, however, there was at first no more than a fibrin clot with very few cells, but by two weeks, and still more at three weeks, there was evidence of tendon cell division, although there was still no sign of vascularisation. In a very tentative conclusion, while stressing that this was a purely experimental study, he thought that free movement prevented adhesion and stimulated growth of the tendon cells in spite of the lack of vascularisation.

Paralytic scoliosis—*Mr J. I. P. James* (London) had attempted to correlate the distribution of paralysis with the curve patterns seen in paralytic scoliosis. Of 193 cases, divisible into high thoracic, thoracic, thoraco-lumbar, lumbar, combined thoracic and lumbar, and " telescopic " spine, he had chosen the high thoracic, thoraco-lumbar and lumbar for special study. When the onset was before the age of five the curvature usually exceeded 100 degrees, but if delayed till after the age of ten it was usually less than 70 degrees. The high thoracic curve usually started at T.1. Unlike Colonna, he had failed to find any correlation between paralysis of the shoulder girdle or proximal segments of the upper limb in this type of curvature. The erector spinae was usually normal and there was no significant relationship to abdominal paralysis. He had however found almost constant paralysis of the intercostal muscles on the convex side, and in contradistinction to idiopathic curves the ribs were not spread on this side but were crowded and hung almost vertically so that they were obscured by the spine in the antero-posterior radiograph. A cineradiographic film showed the ribs on the concave side moving well on respiration in contrast to those on the convex side. In thoraco-lumbar curves with the apex at T.11 or 12, and in lumbar curves, weakness of the lateral flexors on the convex side was constant. Weakness of the erector spinae, anterior abdominal muscles, psoas, glutei or lower limbs was apparently unrelated. The only other factor he thought significant was gravity. He stressed that on the Medical Research Council scale adequate charting of the strength of the trunk muscles was extremely difficult, and estimating the power of the intercostals obviously impossible by clinical methods. *Mr J. C. Scott* (Oxford) questioned whether the high thoracic curve was seen only in paralytic scoliosis as suggested by Mr James. He believed it could be idiopathic and wondered whether any weakness found in the intercostals was not secondary rather than primary. Histological and electromyographic studies had not proved helpful. Mild paralysis could lead to severely progressive curves, and he suggested that interference with vertebral growth might be a major factor. *Mr Robert Roaf* (Liverpool) agreed on the difficulty of testing trunk musculature. It was hard to differentiate the lateral trunk flexors from the outer fibres of erector spinae. He had found that clinical assessment of the intercostals did not correspond with the electrical findings. *Mr J. I. P. James* agreed that electromyographic studies could be misleading. The whole of an intercostal might not be evenly affected; he had noted at necropsy that the anterior half of the muscle might be wasted while the posterior half was in good condition, or vice versa.

Fractures and dislocations of the cervical spine—*Mr F. C. Durbin* (Exeter) had seen seventy-five cases in ten years; of these, twelve involved the atlas or axis and were excluded from consideration in this paper. The rest occurred chiefly in young active men, road accidents being the commonest cause. When paralysis was absent and when other serious and more obvious injuries were present the lesion was easy to miss. He classified the types of fractures seen, stressing that a fractured spinous process always implied damage to an interspinous ligament. In these injuries, if no dislocation was present, he advised that radiographs in flexion carefully controlled by the surgeon in person should be taken to test the stability of the spine. Over 50 per cent of patients with dislocation suffered injury to the cord or nerve roots. In twenty-six the cord was injured; in fifteen there was a complete quadriplegia, of whom fourteen had died. He recommended gradual reduction by skull traction with Crutchfield ice-tong calipers, which were safe and effective if tightened daily. The four failures of reduction by this method were due to locked facets which needed operative reduction. Simple immobilisation after reduction was not sufficient; recurrence of displacement, as shown by radiographs in flexion, had occurred eight times, three of them even after six months in plaster, and two in association with fracture of a spinous process where no dislocation was present at first. He therefore recommended open fixation by wiring the spinous processes and bone grafting in all such injuries; wiring alone was not sufficient. Union was rapid and protection was required for three months only; there had been no deaths in sixteen operations. *Mr D. Lloyd Griffiths* (Manchester) made a plea for the use of the word " tetraplegia " rather than " quadriplegia." *Mr Roland Barnes* (Glasgow) said wiring both deep and superficial to his grafts gave such stability that only the lightest collar protection was then

necessary. He suspected that unilateral locked facets were always irreducible by traction alone and needed immediate open reduction. *Mr M. S. Brett* (Stanmore) recalled the late Mr V. H. Ellis's instructions on the care required in taking flexion radiographs after cervical injury. At operation he removed the traction at the moment the wires were tightened, whereas Mr Durbin maintained traction for a further three weeks. *Mr Durbin* said this traction was light and used simply to steady the head in the immediate post-operative period.

Some aspects of the problem of treating tuberculosis of bone and joint in East Africa—*Mr W. H. Kirkaldy-Willis* (Nairobi) said that there was developing in the last fifteen years a greater appreciation of the benefits of European medicine, but the country was poor, malnutrition widespread, and the paucity of hospital beds necessitated a rapid turnover which militated against efficient treatment. Tuberculosis spread rapidly and the patients were poor operative risks. Adequate follow-up was extremely difficult. However, the response to chemotherapy was often dramatic. His general principle had been to attempt to kill the tubercle bacilli and prevent further spread by chemotherapy and then at operation to remove all dead tissue and proceed forthwith to fusion or, in the case of the elbow, arthroplasty at the same intervention. In the thoracic spine he used the same approach as for an antero-lateral decompression, cleared out the diseased area, curetted the vertebral bodies and then used the ribs he had removed as a body to body inlay graft. At the elbow he excised the lower end of the humerus and the upper ends of the forearm bones through a posterior incision, aiming to leave a gap of 1–1½ inches without any attempt at remodelling joint surfaces. Prolonged physiotherapy was required. In the hip, after removal of diseased tissue through an anterior Smith-Petersen approach, he rawed the joint surfaces and fixed the joint by a massive staple passing from ilium above the acetabulum to femoral neck or trochanteric region. He showed a film illustrating the results of hip and elbow operations, the latter with a full range of elbow movements being particularly impressive. *The president* said that he had been surprised by the relative lack of bone destruction in cases of long duration he had seen in Jamaica and Uganda, but *Mr Kirkaldy-Willis* did not think this was true of Kenya where both destruction and the response to treatment were rapid.

Stress fractures of the fibula—*Mr M. B. Devas* (London) with *Mr R. Sweetnam* (London). This paper was based on a series of fifty cases seen at a special clinic for athletes. In the literature only thirty-four had been previously reported in the lower two thirds of the fibula, eight of these in children. Seen over a four-year period, their series made up between 2 per cent and 3 per cent of the cases attending a special clinic for athletes. In one case fractures involved both fibulae; left and right side were affected almost equally. All their patients were already in training, and the chief cause appeared to be running on a hard surface. A ten-day history was common, and the usual complaint that of pain behind the ankle. In some the onset was sudden, but in others rather more gradual. A few had noticed local swelling. This was always slightly tender, soft at first and later hard. " Springing " the fibula produced pain. The original diagnosis was made on clinical grounds and *not* from radiographs where the changes were slight even after a month (but all these fractures were confirmed radiologically). A fine crack appeared, often affecting only the lateral cortex, and running upwards, inwards and forwards; the periosteal reaction was very slight. The scar in the bone might remain visible for years. The majority (twenty-nine cases) occurred between 4 and 7 centimetres from the lower end of the bone; the highest seen was 23 centimetres up. Two-thirds had occurred in the winter months, perhaps because at that time official tracks were being saved and training done on roads. They believed that the stress was due to muscle action each time the ball of the foot struck the ground; they demonstrated by special x-ray technique that static contraction of the calf muscles without ankle movement caused the upper two-thirds of the fibula to approximate to the tibia, although the lower part remained unmoved. Patients must stop all sport until free from pain and then take up training gradually. Strapping was useful but plaster definitely contra-indicated. If pain recurred they must again reduce their activities. *Professor Bryan McFarland* (Liverpool) asked whether there had been any recurrences. *Mr P. H. Newman* (London) remarked that he had seen a number of fractures affecting the uppermost third of the fibula in parachutists. *Mr G. K. McKee* (Norwich) had seen one fracture progress to non-union, requiring excision and bone grafting. *Mr Devas* said that no recurrence had been observed. He thought that the stress fractures of the uppermost third of the fibula so commonly reported in the German literature and seen in parachutists in this country, were of a different nature and due to repeated relatively major injuries.

ELECTION OF FELLOWS, MEMBERS AND ASSOCIATES

At a meeting on May 4 the following were elected.

Fellows:

Donal Brooks (London)
A. Naylor (Bradford)
W. H. Kirkaldy-Willis (Nairobi)

Members:

J. H. Annan (Huddersfield)
A. C. Clark (Worcester)
J. J. Commerell (Cape Town)
P. S. London (Birmingham)
J. C. McNeur (Melbourne)
K. L. Marks (Chester)
A. K. Mitting (Stoke-on-Trent)
M. F. Pilcher (London)
A. G. Quinlan (Glasgow)
H. J. Richards (Cardiff)
S. Sacks (Johannesburg)
W. H. Scrase (Birmingham)
W. J. W. Sharrard (Sheffield)
J. H. Shelswell (Southend)
R. H. Simpson (Kilmarnock)
T. V. Simpson (Welkom, South Africa)
A. K. Talwalkar (Bombay)
J. G. Taylor (Norwich)
F. T. Wheeldon (Plymouth)
J. K. Wright (Manchester)

Associates:

P. G. Brady (Dublin)
J. R. Briggs (London)
J. H. Calonje (Cali, Colombia)
H. L. Davies (Birkenhead)
E. R. Griffiths (R.A.F.)
G. Hadfield (Pyrford)
J. R. Hutchinson (Glasgow)
W. G. Kerr (Dar es Salaam)
P. Kilburn (Liverpool)
H. Klein (Oswestry)
D. H. McMillan (Mansfield)
D. A. Macpherson (Glasgow)
C. Manning (London)
O. R. Nicholson (Auckland)
J. F. O'Connor (Limerick)
E. L. S. Robertson (Trinidad)
D. E. Rowling (Kirkcaldy)
A. J. Sinclair (Doncaster)
G. A. Wetherell (Liverpool)
P. R. Wheatley (R.A.M.C.)
J. White (Glasgow)

ROBERT JONES CENTENARY MEETING OF THE BRITISH ORTHOPAEDIC ASSOCIATION

The spring meeting will be held at Oswestry and Shrewsbury on May 9, 10 and 11, 1957. Those wishing to read short papers should submit precis to the Honorary Secretary not later than February 1.

The autumn meeting will take place on October 3, 4 and 5, 1957 at the Royal College of Surgeons of England, London.

ROBERT JONES GOLF CUP

After a morning which was thoroughly wetting from a typical fine Scottish mist, the sun came through at lunch time and for once the Robert Jones Golf Cup was played in delightful weather on the King's Course at Gleneagles Hotel on the afternoon of May 3, 1956. The competition produced the satisfactory entry of twenty-nine enthusiasts and the glorious condition of the course, amid some of the finest scenery in Scotland, ensured an enjoyable afternoon for all who took part. The standard of scoring reflected the excellence of the day and the competition was won by Mr N. J. Blockey of Glasgow who returned a score of 78−10=68. This was a net score of 8 under bogey. The sympathy of most of the competitors was felt for Mr G. P. Arden, of Windsor, who had the excellent score of 86−16=70 to occupy the position of runner-up. A net score of 70 usually wins in this competition.

The Robert Jones Cup was presented to Mr Blockey by the president of the Association, Mr Philip Wiles, at the annual dinner held the following evening.

SCIENTIFIC SOCIETIES AND REGIONAL ORTHOPAEDIC CLUBS

THE BONE AND TOOTH SOCIETY

The Bone and Tooth Society held a Meeting at the Institute of Orthopaedics, London, on January 16, 1956. Dr H. A. Sissons was in the chair.

The stability of collagen in vivo—*Dr L. E. Glynn* stated that collagen exhibited a remarkably high resistance to absorption *in vivo* when compared with other tissues such as muscle, liver or spleen. This stability of collagen probably depended upon several factors. Physiological and pathological reabsorption of collagen, however, implied that these stabilising factors were reversible. In order to elucidate the causes of this resistance the influence upon it of various factors had been studied. These factors included death of the associated fibrocytes, strong alkali extraction, heat denaturation, pretreatment with hyaluronidase, the local implantation of hormones and a local Arthur's reaction. The behaviour of acid-soluble collagen and gelatin had also been investigated.

A modified method for the demonstration of nerves in distearate sections of teeth—*Dr S. L. Rowles* and *Mr E. B. Brain* said that staining of nerves in teeth by the method of Ungewitter had shown considerable variation, depending upon the age of component solutions and the pH of the impregnating solution. The nerve staining properties appeared to be connected with the presence of cyanate formed by

tautomerisation of the urea. A modified method had been evolved in which reduction of the silver concentration had diminished general tissue staining. The impregnating solution had been buffered to pH 5·5–6·5, and cyanate (a heated solution of urea) had been included; the picric acid and mercuric cyanide mixture was omitted. Sections of a variety of teeth fixed in formalin, embedded in distearate and stained by this method illustrated consistent results.

Electron microscope appearance of some connective tissues—*Dr K. Little* described the electron microscope appearance of some connective tissues. Typical electron microscope photographs were shown to illustrate the relationship between various components of connective tissues. In cartilage there was a three-dimensional network of collagen fibrils in the ground substance; whereas in reticulin the network was two-dimensional. Tendon and skin showed collagen in bundles, with ground substance between and around the fibrils. At least three different diameters of collagen fibrils could be distinguished with good evidence that many of them were in the form of hollow tubes. The complete separation of collagen and ground substance was difficult—both the electron microscope and x-ray diffraction could be used to assess whether separation was complete.

Bone pyrophosphate—*Dr H. R. Perkins* presented work by *Dr P. G. Walker* and himself which demonstrated the constant presence of a small proportion of pyrophosphate in bone. Quantitative paper chromatographic techniques had been used to show that various regions of rat and rabbit bones contained phosphorus as pyrophosphate to the extent of 0·3 to 1 per cent of the total phosphorus present. When rachitic rat hypertrophic cartilage was incubated with a calcifying solution containing orthophosphate the deposit contained no pyrophosphate, but if ATP was used as the sole source of phosphorus then the deposits could be shown to contain up to 5 per cent of their phosphorus as pyrophosphate. Analysis of the incubating solution showed that it at no time contained any pyrophosphate, although the ATP was rapidly converted into orthophosphate.

IRISH ORTHOPAEDIC CLUB

The spring meeting was held in Dublin on March 24, 1956.

In the morning the Club visited Dr Steevens's Hospital. *Mr Arthur Chance* showed cases of unusual injuries around the ankle joint and demonstrated cases of central dislocation arthrodesis of the hip. He also showed a case of multiple deformities after tuberculous meningitis.

Mr J. Boyd Dunlop discussed the treatment of hallux valgus in adolescence and also showed an excellent foot splint which could be adapted for various deformities.

Mr J. B. Prendiville showed examples of reconstructive surgery of the hand.

Members of the club were entertained to lunch by the Governors of the Incorporated Orthopaedic Hospital of Ireland, and the afternoon was spent in that hospital. *Mr J. C. Cherry* reviewed his results of Elmslie's operation, and *Professor P. M. A. Fitzgerald* reviewed cases of club foot. *Mr J. Boyd Dunlop* discussed congenital elevation of the little toe.

A demonstration of methods of treatment in the gymnasium was given.

SOUTH-EAST METROPOLITAN REGIONAL ORTHOPAEDIC CLUB

The Club met at King's College Hospital on March 17, 1956.

Among a large variety of cases presented by Mr H. L.-C. Wood and Mr R. C. F. Catterall a series of fractures and fracture-dislocations of the cervical spine, successfully treated by Mr Catterall, produced much interest. Mr Catterall advocated the use of zygomatic traction in preference to orthodox skull calipers. The following cases were selected for discussion.

Monostotic fibrous dysplasia of the tibia—*Mr H. L.-C. Wood* showed this patient, now aged seven. A gross deformity of the bone, with much local heat, had been obvious at the age of two. A boiled cortical bone graft obtained from a cadaver had been inserted within the periosteal sleeve after excision of the diseased area. Incorporation was very slow but ultimate fusion was achieved. Five years later the graft had undergone extensive cystic changes and marked bowing of the tibia was again apparent. Sections removed at the original operation had shown no evidence of neurofibromatosis. The etiology of the condition was discussed by Mr W. D. Coltart, who advocated a further biopsy. Re-operation appeared essential at the present stage and Mr E. A. Lindsay suggested using the opposite fibula as a graft and Mr O. J. Vaughan-Jackson suggested the use of McFarland's by-pass method.

Pyogenic arthritis of the hip in infancy—*Mr H. L.-C. Wood* showed three patients. One had developed a pseudarthrosis after destruction of the femoral head and neck. When seen at the age of ten there was 2½ inches of shortening but the hip was stable. The consensus of opinion was that the shortening would probably not increase greatly from this amount and could, therefore, be best treated by epiphysial stapling on the opposite side a little later. In the second case acute osteomyelitis of the ilium was complicated by much sequestration in the ilium and avascular necrosis of the femoral head.

Both sides of the joint appeared to be revascularising well under conservative treatment. The third child had a subluxation of the hip which had been reduced by traction. Good stability was achieved by a shelf operation, but the radiographic appearances were disappointing.

Congenital absence of the radius—*Mr R. C. F. Catterall* showed two patients, in one of whom both forearms were affected. A spirited discussion took place about the treatment of this deformity during which Mr E. W. Bintcliffe described in detail the method he had used in his very successful treatment of such a patient. He advocated prolonged skeletal traction as a preliminary to any operative measure, to achieve full correction of the soft-tissue contracture on the radial side. Traction was effected by a transfixion pin across the metacarpals with counter-traction from a nail through the upper end of the ulna. Later he used a bone graft consisting of the upper end of the fibula (including its epiphysis) screwed to the shaft of the ulna and combined if necessary with an osteotomy of the ulna to correct bowing. He insisted that the fibular graft must be long enough to be attached well up the ulnar shaft while its epiphysis articulated with the radial side of the carpus. In his patient the fibular epiphysis had continued to grow and he proved by radiography that the epiphysial cartilage remained active in this respect. Correction had been well maintained. Mr Coltart pointed out that many people with this congenital deformity achieved surprising efficiency even if untreated or if treatment had failed.

Fractures of the calcaneum—*Mr M. J. Ball* showed four cases with gross involvement of the subtalar joint, treated by early arthrodesis of the subtalar and/or mid-tarsal joints. One patient treated twenty-one years ago demonstrated an excellent late result after subtalar arthrodesis. One patient, a policeman with involvement of both joints, had an immediate triple arthrodesis and was able to return to light duties within six months. Another patient with mid-tarsal arthrodesis only had returned to work in six months.

Mr J. E. Buck and *Mr R. H. Sewell* both advocated early operation if joint damage was severe, but *Mr O. J. Vaughan-Jackson* favoured conservative treatment and maintained that the functional result could seldom be related to the radiological evidence of joint damage. In describing his routine of treatment Mr Sewell maintained that mid-tarsal arthrodesis was seldom needed. Subtalar arthrodesis alone resulted in surprisingly little loss of movement and could be performed simply through a posterior approach. In his series only three out of fifty patients had fractures into the calcaneo-cuboid joint.

Crushed leg with anuria—*Professor C. H. Gray* and *Mr K. Freeman* showed a patient who had sustained a gross crushing injury of the left leg in a road accident. Nearly all the skin was stripped from the thigh and upper half of the calf. There was extensive muscle damage and a comminuted fracture of the femur. There were also lacerations of the perineum and opposite groin. After resuscitation a subtrochanteric amputation was performed and the lacerations of perineum and opposite groin were sutured. Damage to urethra and rectum was excluded. After operation his urinary output was very low and his serum potassium rose rapidly to a high figure. After the failure of various methods of treatment peritoneal dialysis proved successful and urinary excretion returned to normal.

CANADA

CANADIAN ORTHOPAEDIC ASSOCIATION

ELEVENTH ANNUAL MEETING

The eleventh annual meeting of the Canadian Orthopaedic Association was held in Toronto (Ontario) and Montebello (Quebec) from June 25–29, 1955, under the Presidency of Dr John L. McDonald (Toronto).

On June 25 a Clinical Morning was held in the Osler Hall, the Academy of Medicine, in Toronto. Members, guests and wives attended a luncheon at the Royal Canadian Yacht Club as guests of the Canadian orthopaedic members of the Toronto Academy of Medicine, and then travelled to the Seigniory Club, Montebello, Quebec, where the meeting was sponsored by Dr J. Gordon Petrie of Montreal, chairman of the Programme Committee.

The Canadian Orthopaedic Association was pleased to welcome guests from the United Kingdom and the United States, including Mr H. Jackson Burrows (England), Dr and Mrs Ian Fraser (Ireland), Dr and Mrs Beckett Howorth (U.S.A.), Mr J. P. Jackson (England), Mr and Mrs H. H. Langston (England), Dr E. D. Vere Nicoll (U.S.A.), Dr and Mrs L. Weston (U.S.A.), Mr Philip Wiles (England). Canadian guests participating in the programme were: Dr A. W. Carrie (Toronto), Dr Guy Lafond (Montreal), Dr J. R. Lemieux (Quebec City), Dr A. J. Rhodes (Toronto), Dr M. A. Simurda (Newtonbrook), Dr Robert G. Townsend (Montreal), and Dr F. L. VanOrman (Toronto).

Executive sessions—The following officers were elected: President, H. H. Boucher; president-elect, Louis-Philippe Roy; vice-president, A. B. LeMesurier; past-president, John L. McDonald; secretary-treasurer, Ian W. Davidson; editorial secretary, W. B. MacKinnon.

It was reported that Dr Alexander Gibson (Winnipeg) had been chosen as the Special Lecturer in Surgery at the meeting of the Royal College of Physicians and Surgeons of Canada in January of 1955; that Dr W. E. Gallie (Toronto) had been asked to accept an Honorary Degree from the Royal College of Surgeons of Edinburgh, and had been invited to act as Exchange Professor for one week; that Dr R. I. Harris (Toronto) had been chosen as the Sims Commonwealth Professor of Surgery, the first Canadian to be chosen to occupy this important post; and that Dr Cameron S. Allen (Vancouver, B.C.) had been selected to visit Great Britain as Canadian Exchange Fellow from April 20 to mid June 1955.

Presidential address—*Dr John L. McDonald* gave an unusual, interesting and moving address on " the art of medicine," to which it is impossible to do justice in a summary. He referred gratefully to the influence of his teachers, mentioning that he had served as house surgeon to Blundell Bankart and Gordon Taylor and learnt something of the influence of William Osler, Harold Styles, Robert Jones and others. Orthopaedics, in its wide sense, had become the largest and most important branch of surgery. In no branch was it more necessary to cherish the tradition handed down of the dignity of the profession, of service to patients and of the aspirations of our calling.

Prosthetic arthroplasties—*Drs J. E. Bateman, G. F. Pennal, F. P. Dewar* and *D. L. MacIntosh* (Toronto) illustrated, with patients, the wider application of internal prosthetic appliances for occasional use in selected cases, notably partial prosthetic replacement of the head of the humerus and of the tibial condyle in compression injuries.

Hip fusion—*Dr Robert N. Lofthouse* (Hamilton) showed a patient to illustrate his method of arthrodesis of the hip using intramedullary nailing from the ilium to the femur. The advantage claimed was early walking without external fixation.

Encircling metal in fracture repair—*Dr W. O. Stoddard* (Toronto) gave a lantern demonstration of aseptic necrosis of bone and other disadvantages of metal encirclement of bone.

Cruciate ligament repair using meniscus—*Dr Herbert M. Coleman* (Toronto) presented patients with an excellent range of movement and good stability of the knee after repair of a cruciate ligament by a meniscus as advocated by Ian Smillie.

Recurrent dislocation of the elbow—*Dr John C. Kennedy* (London, Ontario) illustrated fully the transfer of the tendon of the biceps to the ulna, to form an active block preventing humero-ulnar dislocation, the method of Reichenheim as modified by King.

Bennett's fracture—*Dr W. R. Harris* (Toronto) advocated open reduction and pin fixation of the detached triangular fragment.

Muscle transposition for paralysis of the deltoid muscle—*Dr W. T. Mustard* (Toronto) gave a preliminary presentation of a patient in whom, four weeks before, he had mobilised pectoralis major proximally and distally to replace the deltoid muscle paralysed in poliomyelitis.

Legg-Perthes disease: genetic aspects and long term follow-up—*Dr R. M. Wansbrough, Dr N. F. Walker* and *Dr A. W. Carrie* (Toronto) reviewed cases of Legg-Perthes disease seen at the Hospital for Sick Children, Toronto, during thirty years, 1920 to 1950. About 130 patients had been re-examined, questioned and x-rayed. From an analysis of the sibs born after the probands in 124 of the pedigrees, the frequency of Legg-Perthes disease was found to be 1/35, compared with the incidence in the general population of 1/20,000—a highly significant difference. The pattern of inheritance was probably incomplete dominance. Dermal patterns recorded some deviation in the foetal growth of affected individuals. The long term follow-up study supported the belief that a reconstitution of the femoral head in its normal anatomical shape gave the best hope of freedom from later arthritis. Various methods of treatment with their end results were carried out. It was suggested that the treatment of choice in unilateral cases was a long Taylor walking caliper on the affected side, with a raised shoe on the opposite side. In bilateral cases a preliminary period of bed rest was necessary to allow one side to heal sufficiently to bear weight. *Dr A. B. LeMesurier* (Toronto), *Dr J. G. Shannon* (Montreal) and *Dr M. Beckett Howorth* (Stamford, Connecticut, U.S.A.) all stressed the difficulty of assessing the results of treatment because of the variable course of the disease. Non weight-bearing ambulant methods were preferred for treatment.

Fractures of the talus—*Dr F. L. VanOrman* (Toronto) and *Dr G. F. Pennal* (Toronto) had studied the blood supply of the talus by injection methods, and reviewed seventy-five clinical cases of fracture. They had concluded that the chief blood supply came from the medial side, particularly through the

deltoid ligament, and that reduction and accurate reposition of fractured fragments, with respect for the blood supply, gave the best prognosis.

Fracture-dislocation of the talus with posterior displacement of the body and avascular necrosis—*Dr Ian W. Davidson* (Sudbury) and *Dr W. J. McCracken* (Hamilton) described several cases of fracture-dislocation of the talus with avascular necrosis, in which open reduction was carried out and the fragments were sutured or screwed. Division of the medial malleolus aided reduction. Weight bearing was permitted as soon as there was evidence of consolidation of the fracture and an early zone of revascularisation beginning beyond it. Some of the patients had now been followed for well over a year and were back at work after a reduction in the total period of disability. Reasonably satisfactory results had been obtained by less complicated methods. *Dr J. A. Leo Walker* (Montreal), discussing both papers on fractures of the talus, agreed that early triple arthrodesis should not be done, but that avascular necrosis demanded protection from weight bearing until the bone had become completely vascularised. In this way the neighbouring joints might escape traumatic arthritis. He suggested the use of a walking caliper to obtain early function with protection from weight bearing. *Dr W. T. Mustard* (Toronto) emphasised that, in triple arthrodesis of the foot, the approach should always be from the lateral aspect to prevent interference with the blood supply to the talus. *Dr G. F. Pennal* (Toronto) emphasised his preference for protecting the avascular talus from early weight bearing and delaying assessment of the final outcome for from five to nine years. *Dr Davidson* considered that in ordinary walking there was very little force transmitted between the body of the talus and the lower end of the tibia. He agreed that some cases might require subtalar fusion later.

Treatment of tuberculosis of the shoulder by resection—*Dr Guy Lafond* (Montreal) said that, during the last twenty years, ninety-one patients with tuberculosis of the shoulder had been treated at the Sacred Heart Hospital. Sixty-one had been treated surgically—twenty-one by arthrodesis and thirty-five by resection. In the twenty-one cases of intended arthrodesis, bony fusion occurred in only nine. He considered resection to be the treatment of choice, especially in women, and that there were few contra-indications to its general use; it was contra-indicated in the adolescent and the elderly; and labourers often complained of instability. *Dr J. Gordon Petrie* (Montreal) pointed out that only four of these thirty-five patients treated by resection had had any antibiotics. The resection of the avascular wall around the lesion might permit antibiotics to be more effective. *Mr H. H. Langston* (Winchester, England) favoured antibiotics followed by arthrodesis. *Dr R. M. Wansbrough* (Toronto) cited examples of tuberculous hips and ankles in children, successfully managed to date by the use of antibiotics. *Mr J. P. Jackson* (Nottingham, England) considered that the series, having been treated before the advent of antibiotics, could not be a reliable guide to present-day problems. *Dr Jos.-Edouard Samson* (Montreal) predicted that in a few years chemotherapy would replace surgery in tuberculosis of the joints, but that in the meantime the preferred treatment was antibiotics and local resection, preserving a mobile joint. In his experience it was most beneficial for women, and often satisfactory for labouring men as well. *Dr J. L. McDonald* (Toronto) commented that the prevention of bone and joint tuberculosis by the pasteurisation of milk had been very effective in the Province of Ontario. *Dr Lafond*, reporting on complications, in his reply to the discussion, stated that eight of the patients had abscesses, five of which healed spontaneously, the others requiring secondary operative procedures; all had come to a satisfactory state of healing.

Experiences in the control of growth discrepancy in the lower extremities—*Dr John C. Kennedy* (London, Ontario) outlined experience with epiphysial stapling in a series of twenty-five cases. Fifteen patients had been followed for over five years. Stapling was used in older patients, eleven to eleven and a half years of age, with a view to arresting growth permanently. In some instances the epiphysis failed to stop growing and the staple became splayed. In only one of five cases in which staples were removed early was it demonstrated that growth began again. Methods in current use for the prediction of growth were reviewed. A method of correction of angular deformities and shortening of the femur was described, in which intramedullary pins were combined with onlay grafts. *Dr C. Laird Wilson* (Montreal) stated that Dr Kennedy had illustrated the fact that compression of the epiphysis would cause closure. He stated that there was evidence to show that traction will also cause epiphysial closure. In his opinion epiphysial erasure was probably better than stapling. He had used the principle of stapling the epiphysis at the convexity of a deformity to obtain a correction. *Dr J. L. McDonald* (Toronto) was not in favour of epiphysial stapling. He believed that there was still a place for lumbar sympathectomy as a leg-lengthening procedure after poliomyelitis. *Dr W. T. Mustard* (Toronto) stated that the force of growth would spread the staples if the patient were too young. If stapling was done later, at a time when it was desirable to oblate the epiphysis, this difficulty did not arise. He recommended a new and stronger staple, made of Vitallium. After sympathectomy of the poliomyelitic limb, apparent lengthening was due to an increased angle of the neck of the femur, and also the development of pelvic obliquity. *Dr Kennedy*, replying, stated that he believed the only

reliable method of increasing blood flow was the method applied by Dr Joseph Janes at the Mayo Clinic, in which an arteriovenous shunt was created, and kept open for two years. Dr Kennedy had had no experience with the Phemister method of epiphysial erasure.

Recurrent posterior dislocation and subluxation of the shoulder—*Dr David L. MacIntosh* (Toronto) said that, although the diagnosis of recurrent posterior subluxation and dislocation of the shoulder from clinical examination was straightforward, none the less it was usually missed altogether, or mistaken for anterior dislocation or subluxation. Thirteen cases were reported, and illustrations were presented to show the posterior position of the head on clinical and radiographic examination. In some instances contraction of the deltoid was sufficient to bring the head over the posterior glenoidal rim. The dislocation occurred with the arm in abduction, medial rotation and forward flexion. The method of repair varied with the findings in the individual case. *Dr J. C. Kennedy* (London, Ontario) suggested that many of these cases were not seen in the stage of acute dislocation, so that the diagnosis between recurrent posterior or anterior dislocation was more difficult. In one case in which the infraspinatus muscle was plicated, recurrence occurred and the Gallie posterior sling method was used.

Experience with recurrent dislocation of the shoulder—*Dr H. F. Moseley* (Montreal) said that a follow-up study was being made of the patients operated on at the Royal Victoria Hospital during 1939–54. During this time there had been a change from the Nicola to the Bankart operation, and then to a modification of this using a Vitallium rim. The essential lesions found on radiological examination and at operation were reviewed. The postero-lateral defect in the humeral head seemed to persist for years after the operation. Measurements of the shoulder capacity suggested that patients with a large anterior subscapular recess required some deepening of the glenoid in front to prevent recurrence. *Dr John C. Kennedy* (London, Ontario) agreed that the Vitallium rim would be useful in those cases with a large anterior recess, and suggested that measurement of the joint capacity might be a valuable contribution, more so if control patients were used rather than cadavers.

Present concepts on the treatment of congenital club foot—*Dr J. Calixe Favreau* (Montreal) based his concepts on a study of three thousand cases of congenital club foot treated by him at the St Justine Hospital, Montreal. The degree of deformity was most variable and did not necessarily indicate the prognosis. Tendency to recurrence was still an obscure problem related to physio-pathological factors which still upset principles and prognosis. There was not yet only one good method for the conservative treatment of congenital club foot; but no method was good unless definite principles were well known and accepted. Often conservative treatment must be completed by surgical procedures, amongst which extensive capsulotomy of the ankle and mid-tarsal joints gave excellent results. The method used was a combination of plaster techniques and the Denis Browne splint, as modified by Dr Alexander Gibson, at various stages in correction. *Dr Gerald L. Burke* (Vancouver) suggested that the term " talipes equinovarus " was inadequate and suggested " pes tortis congenitalis " as an alternative. When asked how early to put the splints on, Dr Burke replied, " in a breech delivery we will apply them before the birth of the after-coming head." *Dr R. G. Townsend* (Calgary) emphasised the greater difficulty of dealing with stiff recurrent club feet.

Chondromalacia of the patella—*Mr Philip Wiles* (London, England). This paper appeared, amplified, in the Thomas Fairbank Birthday Volume of the *Journal of Bone and Joint Surgery* in February 1956.

Report of the Canadian Exchange Fellow to Great Britain—*Dr Cameron S. Allen* (Vancouver). Dr Allen thanked the members of the Canadian Orthopaedic Association for the wonderful opportunity of having been an Exchange Fellow to the British Isles. It was a marvellous trip highlighted by the peerless British hospitality. The tour was exceptionally well organised and the members enjoyed many aspects of the trip, which included the operation lists, the clinical programmes and the social functions.

Excellent research work in basic orthopaedic surgery was in progress at many centres. At the Royal College of Surgeons of England work was being done with the electron microscope, particularly in the study of nerve structure. Biochemical work was also being done on isolating the 17-ketosteroids from the urine. Detailed studies of vascular anatomy of various organs yielded interesting specimens. In Edinburgh work was proceeding on mucopolysaccharides—an attempt to determine the exact structure of chondroitin sulphuric acid. The healing of fractures was being studied by radioactive isotopes. In Oxford study of the circulation of various bones was in progress, including that of the scaphoid and the head of the femur. At the Institute of Orthopaedics, Royal National Orthopaedic Hospital, correlation between muscles paralysed with poliomyelitis and damaged nerve cells was forwarding the knowledge of the anatomy of the spinal cord. At Birmingham blood volume in acute injuries was under study.

In the treatment of tuberculosis a direct attack on the tuberculous foci, even in Pott's disease, was advocated. Biopsies were frequently resorted to for diagnosis. At Sheffield, in a paraplegic

centre, open operation with Wilson plate and bone graft were done for traumatic fracture-dislocations. In the treatment of congenital dislocation of the hips arthrograms were frequently used, and these resulted in more open reductions, in which the limbus only was excised and the hip then reduced and put up in marked medial rotation in plaster. Before this, traction was placed on the leg for about three weeks. Three weeks after the open reduction, osteotomy was done below the greater trochanter and the leg rotated laterally at least 90 degrees. At Edinburgh a series of thirty patients with leg lengthening operations was presented, with a gain in length of one-half to three and a quarter inches. These showed no serious complications from ischaemia or nerve damage. A great variety of operations for osteoarthritis of the hip were seen, including the cup arthroplasty, prosthesis, McMurray osteotomy, Charnley stabilisation, and Girdlestone excision of the hip, and also arthrodesis of the hip, by the Watson-Jones method. Excellent results were presented from these various methods.

Many of the hospitals had a brace shop. The rehabilitation of orthopaedic patients was very completely carried out and special retraining schools were provided.

Dr Allen thought that there was not any difference in the basic approach to orthopaedics in America and in Great Britain. It was stimulating and rewarding to meet famous and great orthopaedic men, and the opportunity of travelling with the four young American orthopaedic surgeons was an outstanding one.

Recent developments in vaccination against poliomyelitis—*A. J. Rhodes* (Toronto) said that until a few years ago the only method of studying poliomyelitis in the laboratory was by the inoculation of monkeys. In 1949 the study of poliomyelitis was revolutionised by the discovery of Enders, Wells and Robbins that the virus could be grown in human tissue cultures. This led the way to the development of a vaccine for protection against the infection. Earlier experience with vaccines containing live virus had resulted in some disasters and discontinuation of the use of the vaccine. In 1938 Lansing demonstrated that the Lansing virus could be transmitted to mice, and it became possible to measure accurately poliomyelitis viruses in suspensions and poliomyelitis antibody. Dr Isabel Morgan injected live Lansing virus intramuscularly into monkeys, and noted that the antibody played the major role in determining resistance to infection. Animals which developed antibody levels of approximately 1 : 1000 after vaccination were resistant when inoculated with Lansing virus into the brain. Bodian and others showed that poliomyelitis antibody inoculated into monkeys protected against virus given intramuscularly, subcutaneously, intravenously and even into the brain. The work of Hammon showed that gamma globulin could prevent a certain percentage of cases of paralytic poliomyelitis. Bodian and Horstman demonstrated that during the incubation period in poliomyelitis there was a period of generalised virus invasion or viremia, both in animals and man. After circulating in the blood for up to five days, virus might settle in the central nervous system. Therefore, the time to attack poliomyelitis infection was during this stage of viremia; experimental evidence suggested that, if antibody were present in the blood, viremia would not occur, and so there would be no invasion of the central nervous system.

Three distinct types of poliomyelitis virus had been found. Dr Leon Farrell and her associates in the Connaught Medical Research Laboratories devised a method whereby poliomyelitis virus was grown in minced monkey kidney tissue in large diphtheria toxin bottles in approximately 750 cubic centimetres of synthetic Medium 199. Jonas E. Salk was entrusted with the task of devising a method of destroying poliomyelitis virus in such a way that antigenicity was still retained. He devised a method of killing the virus by means of formalin, and showed that it produced antibody levels in approximately one in one hundred. When the antibody level declined it could be stimulated to high levels by a booster dose not less than seven months after the primary course. The extensive field trials in 1954 showed that there were only between a fifth and a tenth as many cases of paralytic poliomyelitis in the vaccinated as in the control groups for Types 2 and 3 infection. In the Type 1 there were approximately one-third as many cases of poliomyelitis in the vaccinated group as in the controls. It appeared that the preservative (Merthiolate) added to the vaccine depressed the antigenicity of Type 1 component to the vaccine. This defect was remedied in vaccines in use for 1955.

In Canada in 1955 over 800,000 children, mostly Grades 1, 2 and 3, had received two inoculations of Salk-type vaccine. It was intended that these children should receive a third or booster inoculation several months later. The programme, sponsored by the Federal and Provincial Governments, would later be expanded. There had been no authenticated instance of paralytic poliomyelitis attributable to the vaccination. The Salk vaccine remained under study, and, should it give disappointing protection, it would be necessary to explore the possibility of using an attenuated strain of virus. The future in regard to the prevention of poliomyelitis was full of hope.

Diastematomyelia—*Dr W. W. Ollerhead* (London, Ontario) said that diastematomyelia signified an abnormal cleft or division of the spinal cord or cauda equina: a discrete bony spicule projected backwards from the posterior surfaces of a vertebral body through the middle of the spinal canal,

so as to divide the meninges into two compartments and transfix the spinal cord or cauda equina. An embryological explanation was that abberant mesodermal cells protruded into the neural canal on its ventral surface instead of becoming arranged around it during the organisation of the neural tube from the primitive neuro-ectoderm. Early in embryological development the spinal cord and vertebral column were of equal length. The vertebral column grew more rapidly, and there was an apparent migration cranially of the cord. At birth the conus medullaris was found opposite the third or fourth lumbar vertebra, and, by the time growth had ceased, it lay opposite the first lumbar or slightly higher. If the cord was transfixed by a bony spur during this growth period, damage to nerve tissue was likely to result. The patients were brought because of impaired function of the lower extremities. Difficulty in walking or learning to walk was the most common complaint. Impaired function of vesical or rectal sphincters, muscle weakness and imbalance, atrophy, unequal length of extremities or foot deformity might occur. Sensory changes and loss of tendon reflexes might be present. There might be cutaneous abnormalities, such as tufts of hair, dimpling of the skin, cutaneous blood vessel malformation, or fatty tumours. The condition might be present at any site from the fifth cervical to the fourth lumbar vertebra. The antero-posterior radiographs showed a spot of increased density in the centre of the spinal canal, characteristically in an area of spina bifida extending over one or more segments. Myelography would demonstrate a bifid dural sac, the opaque medium being separated into two columns by the septum. Operation consisted in removal of the bony spur and reconstructing the dural sac to form a single tube. The aim was to prevent further damage to the central nervous system, but there might be some improvement. A case was presented in a girl first seen at the age of five and a half months with " a lump in the back and a small left leg." She was watched until the age of four and a half years, when her leg inequality was increasing and scoliosis was developing, and her bowels were constipated. The bony spicule was recognised. Myelography confirmed the diagnosis, and operation caused marked symptomatic improvement.

Histopathology of Morton's toe—*Dr J. Paul Roger* (Quebec City) and *Dr J. R. Lemieux* (Quebec City) contended that an anatomical anomaly of the third plantar interdigital artery rendered it vulnerable. Repeated trauma of narrow shoes or even walking compressed the artery between the two metatarsals. Compression of the vasa-vasorum affected all elements of the artery, and consequently the interdigital nerve, which in turn degenerated, and became replaced by fibrous tissue. Degeneration of the axis cylinder and the sheath of Schwann were followed by fibrous hyperplasia originating from the endoneurium and perineurium. Morton's disease was defined as a progressive and chronic lesion caused by a simultaneous post-traumatic ischaemia of the third interdigital plantar artery and the corresponding nerve, and a consequent degenerative fibrous hyperplasia of both these elements.

Behaviour of metals in tissue—*Dr Gerald L. Burke* (Vancouver) produced evidence that, quite apart from electrolytic changes, all metals available for use undergo corrosive changes from surface oxidation.

Tears of the fibrocartilaginous disc at the wrist—*Dr Herbert M. Coleman* (Toronto) recalled that, although injury to the fibrocartilaginous disc at the wrist had long been considered a cause of disability after a Colles's fracture, since Petit first suggested it in 1726, little had been published in confirmation. The mechanism of injury was severe trauma produced by hyperextension rotational strain at the wrist. This was followed by a painful " click " in the wrist joint and tenderness over the inferior radio-ulnar joint. The pain was aggravated by use, particularly rotational strain, and had been severe enough to keep workmen from their regular employment. Four cases were presented in which tears of the disc were demonstrated at operation. They were illustrated by a moving picture, the mechanism of the tear being demonstrated in the cadaver.

Early functional results of arthroplasties on the hip—*Dr F. P. Dewar* (Toronto) and *Dr M. A. Simurda* (Newtonbrook) had surveyed 120 arthroplasties of the hip at the Toronto General Hospital over a five-year period. The average interval since operation was two years, the least six months. The series included operations using the Smith-Petersen cup, the Judet (acrylic) prosthesis and the Moore (Vitallium) intramedullary prosthesis. The diseases for which the operations had been performed included neuropathic joint disease, arthritis, fractures complicated by non-union and avascular necrosis, and fresh fractures. The purpose of the follow-up was to assess the resulting hip function, the amelioration of symptoms and the durability of the reconstruction. On the basis of mobility index and patients' assessment, a large percentage of these cases were satisfactory. *Dr Alexander Gibson* (Winnipeg) considered that an assessment less than five years after operation was of little value and that the group was too heterogeneous for conclusions.

A universal foot support—*Dr Henry Funk* (Winnipeg), recalling that a rigid support is often fitted to a supple foot, described the construction of a support of sponge rubber and belt leather to fit the individual patient.

Local excision of potentially malignant neoplasms—*Mr H. Jackson Burrows* (London, England) gave a preliminary presentation, with lantern slides, of radical excision of potentially malignant endosteal neoplasms and their replacement by prostheses in collaboration with Dr J. T. Scales. The cases, of which the first had been observed for five years after operation, were very carefully selected and were such that the alternative treatment would have been amputation.

INDIA

ASSOCIATION OF SURGEONS OF INDIA

At a meeting of the Association of Surgeons of India held in December 1955 it was decided to form an orthopaedic section, and the first Regional Conference of the Section was held in April 1956. This was attended by a representative contingent of orthopaedic surgeons from different parts of India.

INTERNATIONAL SOCIETIES

INTERNATIONAL CONGRESS ON RHEUMATIC DISEASES

The Ninth International Congress on Rheumatic Diseases will be held at Toronto, Ontario, Canada, from June 23–28, 1957. This quadrennial function of La Ligue Internationale contre le Rhumatisme will be held under the auspices of the Canadian Rheumatism Association.

The Programme Committee invites contributions to the scientific programme of the Congress and is anxious to receive reports on current clinical or basic research dealing with any aspect of the rheumatic diseases.

Those offering papers for consideration should submit a 200 to 300 word abstract not later than January 1, 1957. Abstracts should be submitted in triplicate in the language in which the paper is to be read. If an abstract is submitted in a language other than English, it will be helpful, though not essential, if it can be accompanied by an English translation.

All correspondence should be directed to the Ninth International Congress on Rheumatic Diseases, Post Office Box 237, Terminal " A," Toronto, Ontario, Canada.

Book Reviews

MANUAL OF HAND INJURIES. By H. Minor NICHOLS, M.D., Clinical Instructor in Surgery, University of Oregon Medical School, Portland. 9×6 in. Pp. 352, with 180 figures. Index. 1955. Chicago: The Year Book Publishers Inc. London: Interscience Publishers Ltd. Price 72s.

During the past twenty years an increasing number of surgeons in America have specialised in the surgery of the hand. Kanavel and Stirling Bunnell have set a very high standard in this new speciality. H. Minor Nichols's Manual of Hand Injuries maintains this quality of writing. His descriptions of anatomy are short and practical and accompanied by clear illustrations. The chapter on general technique contains sound principles of the management of these injuries. The chapter on minor injuries is full of useful and practical advice for all medical practitioners. It is interesting to find that the myth about venous compression and Volkmann's contracture still persists in American

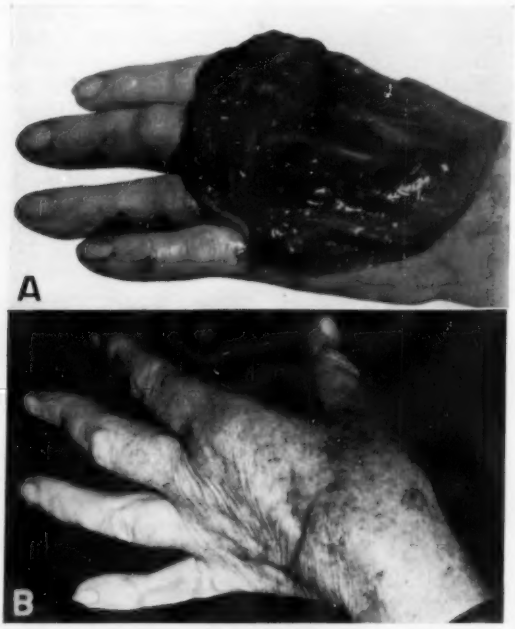


FIG. 63

A: Avulsion of skin of back of hand from auto accident. Excision of subcutaneous tissues and replacement of skin as graft resulted in full function (B).

writings. The remarks about burns are sound and up to date, and there are full and concise descriptions of the plastic surgery indications and techniques. There is an excellent discussion on the problem of avulsed skin flaps, and the author is quite certain that the chances of recovery are better if the flaps are detached, denuded of fat and then sewn back into position (Fig. 63). Photographs of extensive, complicated injuries are accompanied by explanatory line drawings which make the illustrations most effective. The chapter on infections is disappointing. There is no advance on the treatment described by Kanavel and one regrets to see the recommendation of such out-dated treatment as moist dressings and "fish mouth" incisions. The only other fault to be found is the recommendation that the fluoroscope is used in checking the position of a fracture. Aside from these two faults the book is so informative and so well produced that it should be considered an essential for the library of surgeons dealing with accidents.—L. W. PLEWES.

OUTLINE OF ORTHOPAEDICS. By John Crawford ADAMS, M.D., F.R.C.S., Orthopaedic Surgeon, St Mary's Hospital, London, and St Vincent's Hospital, Pinner; Assistant British Editor, *Journal of Bone and Joint Surgery*. 8½×5½ in. Pp. viii+423, with 299 figures. Index. 1956. Edinburgh and London: E. & S. Livingstone Ltd. Price 32s. 6d.

The General Medical Council prescribes for the medical student " instruction " in orthopaedics " directed to the attainment of sufficient knowledge to ensure familiarity with common conditions, their recognition and treatment." Those who examine him demand a good deal more than this, and so, later on, will his patients. The thinking student prefers education to " instruction," and so does the thinking teacher. In perhaps most medical schools the facilities for orthopaedics are still more suited to an obscure and upstart cult than to a major part of surgical practice and a splendid exemplification of applied physiology. Consequently there has been an insistent demand for a students' text-book, if possible of the brevity demanded by the grossly overcrowded course. It is surprising how few text-books of orthopaedics there are, even allowing that the subject is difficult to present and its practitioners very busy people.

Mr Adams's book is intended primarily for undergraduate students. There is no branch of medicine that lends itself better to the practice of observation and clinical examination, and Mr Adams has wisely opened his book, after a brief historical introduction, with a chapter on Clinical Methods. This chapter is a masterpiece. He has condensed into nineteen pages a detailed account of physical examination, a brief but adequate reference to special investigations and a summary of the methods of treatment for orthopaedic disorders. Brevity has sometimes been carried too far. Manipulation is advocated for a stiff joint and for deformity—just the conditions in which manipulation is difficult, undramatic and dangerous; Paget's " cases which bone setters cure " are left out. The opportunity has been missed of a physiological approach to physiotherapy, which would make the student think in terms of function and enable him to apply this treatment intelligently and safely. On the other hand the approach to " operative treatment " is a model; here is the opening paragraph: " The chief essential of any treatment is that it should not make the patient worse than he was before he submitted himself to it. This is so obvious that the statement may sound almost absurd. Yet it is unfortunately true that a disturbing number of operations carried out for orthopaedic conditions do in fact cause more harm than good for one reason or another. Hence the selection of cases for operation, the choice of the most appropriate operation in given circumstances, the technical performance of the operation, and the post-operative management are matters of the highest importance, and they call for a high degree of judgment and skill. Herein lies much of the fascination of orthopaedic surgery."

Between the opening chapter and the topographical chapters the author has wisely inserted a general survey of orthopaedic disorders. This is most valuable and saves a great deal of repetition in the topographical section. This and the later chapters, however, are necessarily synoptic and do not reach the high standard of the first chapter. If they had the life, the precision, the logic and the wisdom of their forerunner the book would be too long—but would its fare take longer to consume and digest than the tablet ration here presented?

There are, as in all first editions, slips that will need correction in the further editions that will certainly be demanded. For instance the modern decline in tuberculous arthritis in Great Britain is confidently ascribed to general pasteurisation of milk; whereas the chief culprit has been the human bacillus ever since a distinction was possible.

The illustrations are excellent. Many resemble blackboard diagrams and are the work of the author, who has a rare knack for bringing out the essential points. The radiographs are well chosen and well reproduced.

Mr Adams is to be congratulated on this book; its success is assured. How he managed to write it, in spite of the enormous burden of Journal work that he sustains, is a complete mystery.—H. Jackson BURROWS.

RHUMATOLOGIE CLINIQUE. By Pierre P. RAVAULT, Professeur de Clinique Médicale à la Faculté de Lyon, Médecin de l'Hôpital E. Herriot; and Georges VIGNON, Professeur agrégé à la Faculté de Lyon, Médecin des Hôpitaux. 10×7 in. Pp. 604, with 180 figures. Index. 1956. Paris: Masson et Cie, Editeurs. Price, in paper cover, 4.000 fr., in cloth boards, 4.600 fr.

In France rheumatology comprises in the main the study and treatment of *arthritis* (inflammatory rheumatism) and *arthrosis* (degenerative rheumatism). The present work which comes from the Faculté de Lyon provides a notable exposition of the French outlook. Its language is easily read by an Englishman, perhaps because it is written for the postgraduate student rather than as an advanced text-book. Nevertheless it is a large work with relatively few illustrations, and it deals adequately with every aspect of the subject, including the most modern work upon adrenal hormones.

The French influence in the field of the rheumatic diseases has been notable. It is well stated here; furthermore recognition is given to work in other countries, though strangely the bibliographies are almost entirely French. While each section gives a discussion of surgical treatment, the viewpoint expressed is that of the physician. In the treatment of osteoarthritis of the hip, osteotomy is thought of only to correct deformity and to relieve pain by changing weight-bearing alignment: it is regarded as palliative and not very durable. There is, however, recognition of the value of arthrodesis, and, while due note is given to the advances made in arthroplasty by Smith-Petersen and by Judet, yet one senses an attitude of caution.

Rheumatology in France, as in Britain, appears to include almost every painful condition of the locomotor system, whether or not it comes strictly within the definition at the beginning of this review.—Norman CAPENER.

RECONSTRUCTION SURGERY AND TRAUMATOLOGY. Edited by M. LANGE and twenty other editors. Volume III. $9\frac{3}{4} \times 7$ in. Pp. 181, with 55 figures. 1956. Basel/New York: S. Karger. Price S.Fr. 32.

The first two volumes of this series were published in 1953 and 1954 and were concerned almost entirely with trauma. The third volume now produced deals solely with tuberculosis of bone and joint. There are six articles, three in German, two in English and one in French. E. Sorrel and Mme Sorrel-Déjerine (Paris) conclude that the introduction of antibiotics has improved the accepted methods of treatment of tuberculosis in bone and joint, but has not displaced any of them. General treatment accompanied by immobilisation in children, and resection or arthrodesis in adults, are still essential for good results. S. Orell (Stockholm) discusses spinal tuberculosis in relation to antibiotic therapy and finds a considerable reduction in morbidity rate as a result of its use combined with more radical surgery. S. Colombani (Bologna) writes enthusiastically in favour of antibiotics. He states that incomplete removal of the tuberculous focus can be curative in the presence of antibiotic therapy. Furthermore, it is now possible and safe to preserve mobility in a diseased joint. De la Sierra Cano (Santander, Spain) concludes that great advances have been made in the treatment and rehabilitation of patients with skeletal tuberculosis since the advent of antibiotics. He favours local excision under specific cover, but insists on general treatment as well. M. Horvat (Rovinj, Jugoslavia) points out that the mortality from skeletal tuberculosis has decreased because of the beneficial effect of chemotherapeutic and antibiotic agents on sinuses. He agrees with the other authors that both conservative treatment in children and operative treatment in adults have been improved with the use of these agents. M. Lange and G. Glogowski (Munich) favour the use of INH and streptomycin, and give their views on the dosage and indications and on the possible modifications to established operative procedures.—L. W. PLEWES.

ARTHROPLASTY. By St J. D. BUXTON, M.B., F.R.C.S., Consulting Orthopaedic Surgeon and Emeritus Lecturer in Orthopaedics to the Medical School, King's College Hospital; Past President, British Orthopaedic Association; formerly Consulting Orthopaedic Surgeon to the Army. $8 \times 5\frac{1}{2}$ in. Pp. viii+126, with 2 plates and 43 figures. Record charts, bibliography, index. 1955. London: Pitman Medical Publishing Co. Ltd. Price 30s.

This slim work deals with arthroplasty from most points of view—history, principles, indications, details of operation and subsequent treatment, difficulties, complications and results. The author speaks from his own extensive experience, and with authority. The illustrations are clear and the bibliography is extensive.

The joints of both upper and lower limb are considered. The difficult problems of the hip receive most attention, especially replacement of the femoral head by a plastic prosthesis, an operation now widely discredited. Of fifty-six consecutive patients subjected to this operation, forty-four have been kept under observation for twelve to thirty-two months. This has allowed an admirable review of the early effects with special reference to the radiological changes. The advantages are stressed of delaying any major arthroplasty for some days after the patient's admission to hospital. The management of the patient before and after operation is rightly described in much detail.

Inevitably there are views with which one would take issue. Total removal of the first metatarsal head for hallux valgus or hallux rigidus is condoned. It is stated—twice—that plastic replacement of the femoral head after high cervical fractures is easier than nailing; for some surgeons it probably is, but is it ever easier for the patient?

The author looks to a bright future for arthroplasty, even in the hip, which has provided so many disappointments.—H. Jackson BURROWS.

CARDIOVASCULAR INNERVATION. By G. A. G. MITCHELL, *O.B.E.*, *T.D.*, M.B., Ch.M., D.Sc., Professor of Anatomy, University of Manchester. Foreword by Sir Geoffrey JEFFERSON, *C.B.E.*, M.S., M.Ch., M.Sc., LL.D., F.R.C.P., F.R.C.S., F.A.C.S., F.R.S., Emeritus Professor of Neurological Surgery, University of Manchester. 10×7¼ in. Pp. xii+356, with 217 figures, many in colour. Index. 1956. Edinburgh and London: E. & S. Livingstone Ltd. Price 55s.

It must be well over twenty years since Professor Mitchell thought of writing a book on the innervation of the heart and blood vessels. Sir James Learmonth, with whom he was then associated in Aberdeen, suggested to him that he should first write an up-to-date and balanced account of the central and peripheral parts of the autonomic nervous system, and the result was the classic work *The Anatomy of the Autonomic Nervous System* which appeared in 1953. Having discharged this duty he returned to his original objective, and has now placed the innervation of the heart and blood vessels in the correct relationship to the whole of the autonomic system.

One might expect a volume which deals with only a part of the autonomic nervous system to be somewhat smaller than the one which included the central connections and peripheral distribution of both the sympathetic and parasympathetic systems, yet the two books are of practically the same size because Professor Mitchell has gone into much greater detail in his description of the cardiovascular nerves, especially in their terminal distribution, and the new volume contains over seventy more illustrations. When comparing the two books one cannot help noticing how many of the illustrations are identical, and there are sections of the text which are reproduced word for word—yet this is not intended as adverse criticism, for, after all, both books are dealing to a great extent with the same subject and differ only in the emphasis that is placed upon certain portions of the same nervous system.

Throughout the book one is impressed not only by the painstaking accuracy of Professor Mitchell's own dissections but also by his careful and discriminating search of the literature which affords us a most valuable summary of the researches of other anatomists, physiologists and surgeons who have worked in this field. In his attempt to be scrupulously fair to others he has occasionally sacrificed some of the clarity that distinguishes his accounts of his own investigations—an example of this is shown in Figure 35 which illustrates only "approximate" segmental levels of sympathetic representation in the spinal cord.

The production of the book and the quality of the drawings and photographs which illustrate it are first rate and greatly enhance the value of Professor Mitchell's work, which will be welcomed by clinicians and also by their more scientific colleagues as a rich and reliable source of information on a system that is commonly regarded as being rather out of reach. The ease with which this book can be used as a work of reference goes far to dispel this idea and to make cardiovascular innervation more comprehensible.—J. Paterson Ross.

THE DIAGNOSIS AND TREATMENT OF POSTURAL DEFECTS. By Winthrop Morgan PHELPS, M.D., Medical Director, Children's Rehabilitation Institute for Cerebral Palsy, Baltimore; Robert J. H. KIPHUTH, M.P.E., Professor of Physical Education, Yale University; and Charles Weer GOFF, M.D., Associate Clinical Professor of Orthopaedic Surgery and Lecturer in Anatomy, Yale University School of Medicine. Second edition. 9½×6¼ in. Pp. xi+190, with 87 figures. Index. 1956. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications. Price 47s. 6d.

There is perhaps no greater divergence between British and American thought, in so far as orthopaedics are concerned, than in the field of posture. British views are based on the assumption that the posture of the body is regulated by the central nervous system; Americans, following the older Germanic school, tend to stress mechanical factors and concern themselves more with muscle power. Hence the former, when treating postural defects, emphasise the importance of re-educating postural reflexes—balance rather than strength, whilst the latter devote themselves to strengthening exercises—strength must dominate. In practice, the patients of both schools may be given similar exercises, but the motivating ideas are different.

This book, which is designed for physical training instructors and athletic coaches rather than for orthopaedic surgeons, gives an excellent presentation of the American view. It opens with an account of what are considered to be evolutionary influences on the posture of man. Next follows a description of the changes in posture that take place during growth, and of normal adult posture; this section is the result of an extensive and painstaking study and is really first class. The final chapters on body mechanics, postural examination and corrective exercises for strengthening fairly represent the mechanical outlook. The book is generously illustrated with photographs and is well produced.—Philip WILES.

ROENTGEN INTERPRETATION OF FRACTURES AND DISLOCATIONS. By Joseph LEVITIN, M.D., Chief, Department of Radiology, Mount Zion Hospital, San Francisco, California; and Ben COLLOFF, M.D., Associate Chief, Department of Orthopaedic Surgery, Mount Zion Hospital, San Francisco, California. 9¼ × 6¼ in. Pp. xiv + 265, with 258 figures. Index. 1956. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications. Price 55s. 6d.

This book is described as an aid to the roentgenologist and to those general practitioners who treat fractures and dislocations. As such it is unlikely to have much demand in this country, particularly among orthopaedic surgeons, who are accustomed to interpret their own radiographs. Few surgeons here would agree with the observation on page 179 that " No other procedure in orthopaedic surgery requires as close supervision and co-operation between the surgeon and the roentgenologist as does reduction and pinning of hip fractures."

For a book confined to radiographic interpretation there is a remarkable sparsity of radiographs. Those that are included illustrate only the various appliances used in the treatment of fractures of the neck of the femur, and the quality of some of these reproductions is very poor. The text is otherwise widely illustrated by line drawings. These show the common varieties of fractures very clearly. Particularly to be commended are the descriptions of the various types of Pott's fracture, which could prove useful for student teaching. It is a pity that, as radiologists, the authors did not include the interpretation of some of the more difficult fractures: for instance, there is no mention at all of facio-maxillary or skull injuries—the very type of fracture where the co-operation of surgeon and radiologist is often most needed.—J. N. WILSON.

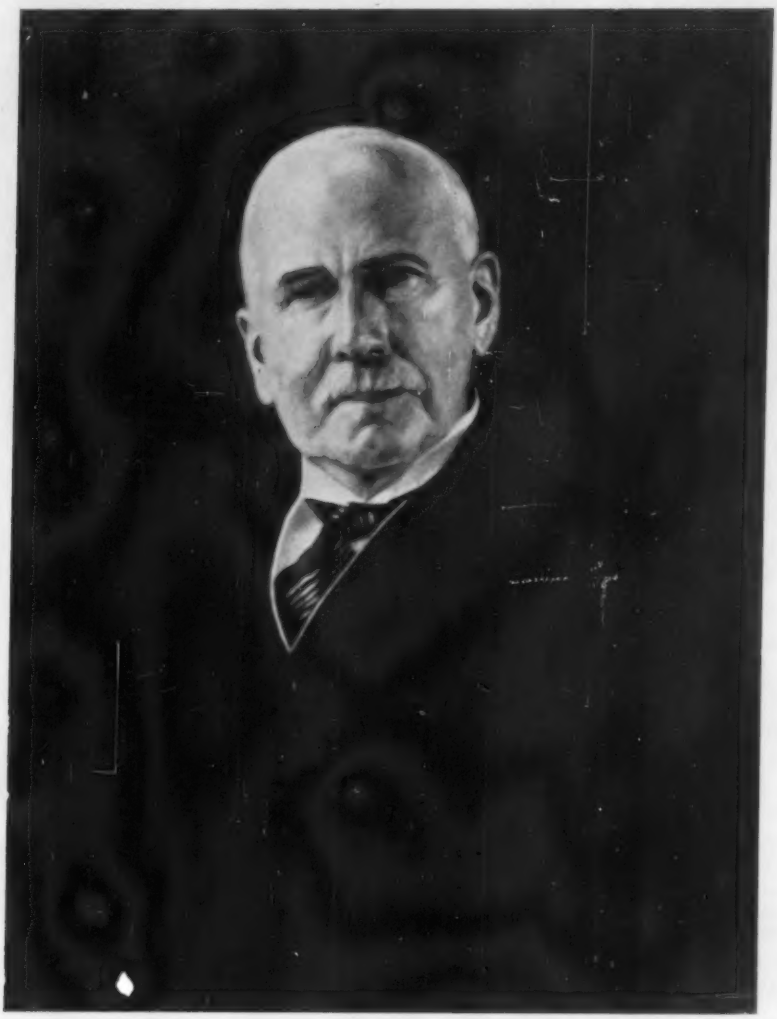
THE CERVICAL SYNDROME. By Ruth JACKSON, M.D., F.A.C.S., Clinical Assistant Professor of Orthopaedic Surgery, Southwestern Medical School of the University of Texas, Dallas. 8¾ × 5¾ in. Pp. viii + 130, with 50 figures. Index. 1956. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications. Price 34s.

This monograph, one of a series in orthopaedics, is concerned with the " cervical syndrome," a term used by the author to embrace a variety of orthopaedic conditions such as the cervical disc syndrome, frozen shoulder, calcareous deposit in the shoulder cuff, epicondylitis, and fibrotic changes in the palmar fascia. Dr Jackson evidently believes, if the reviewer understands her correctly, that these lesions are caused by irritation or compression of the cervical nerve roots in or about the intervertebral foramina. In some of her patients she has demonstrated a narrow space in association with calcareous deposits in the shoulder cuff (Fig. 43), and on this slender evidence argues that there is a relationship between the two. The author is on safer ground when she states that the common cause of root irritation is a spur formation at the lateral intervertebral joints, and not, as commonly supposed, a disc protrusion. Narrowing of the intervertebral foramina by spur formation is best demonstrated by oblique radiographs of the cervical spine. The treatment by traction, physiotherapy and correction of bad posture is fully discussed, and the importance stressed of using a pillow of the correct height and shape whilst sleeping. The monograph is well produced and illustrated, and is of a convenient size.—Roland BARNES.

SIR WILLIAM ARBUTHNOT LANE, BT., an enquiry into the mind and influence of a surgeon. By T. B. LAYTON, *D.S.O.*, M.S., F.R.C.S., Consulting Surgeon Emeritus (Ear and Throat), Guy's Hospital, London. 9 × 6¼ in. Pp. viii + 128, with 9 figures. Index. 1956. Edinburgh and London: E. & S. Livingstone Ltd. Price 21s.

Arbuthnot Lane was one of the most controversial figures of the early part of this century but it must come as a shock to many to read that " Lane initiated nothing. In all that he did he adopted, adapted and extended the work of others." There are two exceptions: his plates used for internal fixation and the " no touch " technique to which his name is still attached. His reputation rests on his work as a pioneer in traumatic surgery, where his skill enabled him to perfect the technique of open reduction and the rigid asepsis made the use of foreign material safe. At this length of time it is difficult to appreciate the resistance of some of his contemporaries to ideas which we have accepted, and in estimating his achievements in asepsis we are apt to forget, for example, that rubber gloves were not available when he started his work.

This biography, written by one of his house surgeons at Guy's, aims at tracing his influence on the surgery of his time rather than giving a factual account of his career, and the author has kept a balance between the high regard in which his bone work is held and the disrepute into which much of his other work has fallen.



After the portrait by Mrs Irma Hardy.

FIG. 8
As we last remember him.

Part of the book is concerned with the influence of his seniors at Guy's on Lane before and after he got on the staff of the Hospital, and so is filled with names and anecdotes which may mean little to younger men trained at other hospitals. But as a whole it is an interesting account of the character and qualities by which Lane reached such a distinguished position among his rivals.—A. T. FRIPP.

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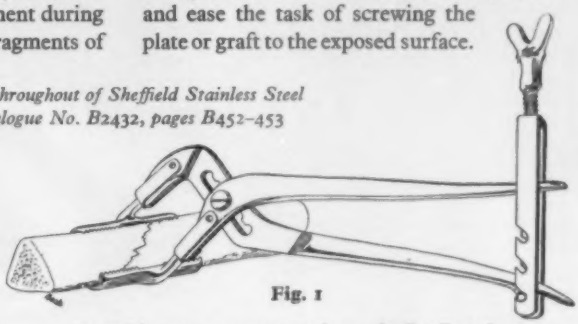

Fig. 1

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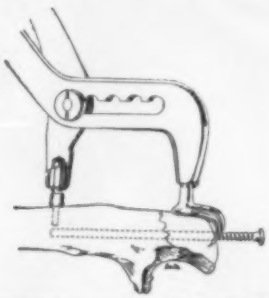

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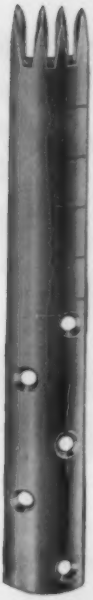

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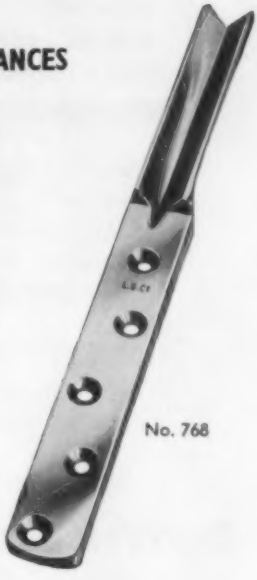

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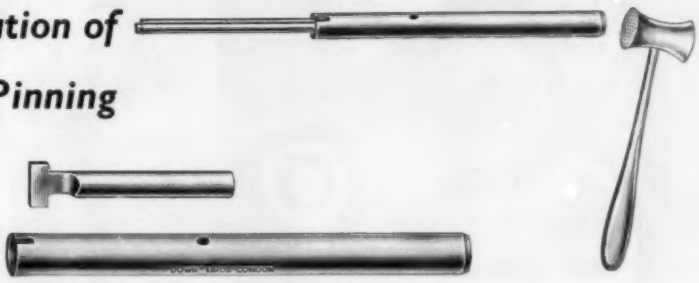

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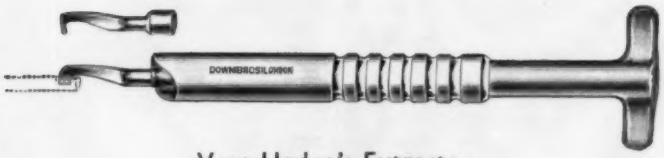

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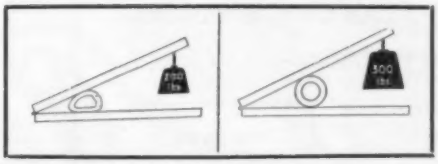

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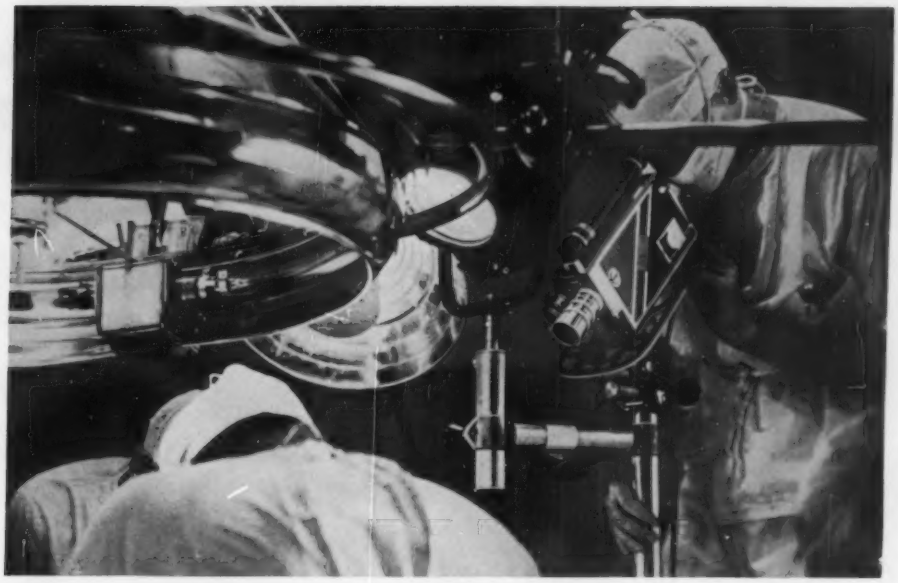

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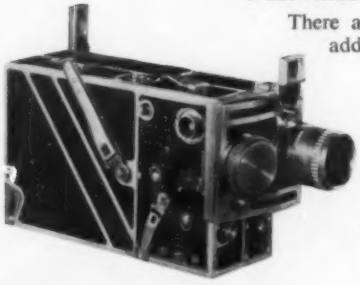

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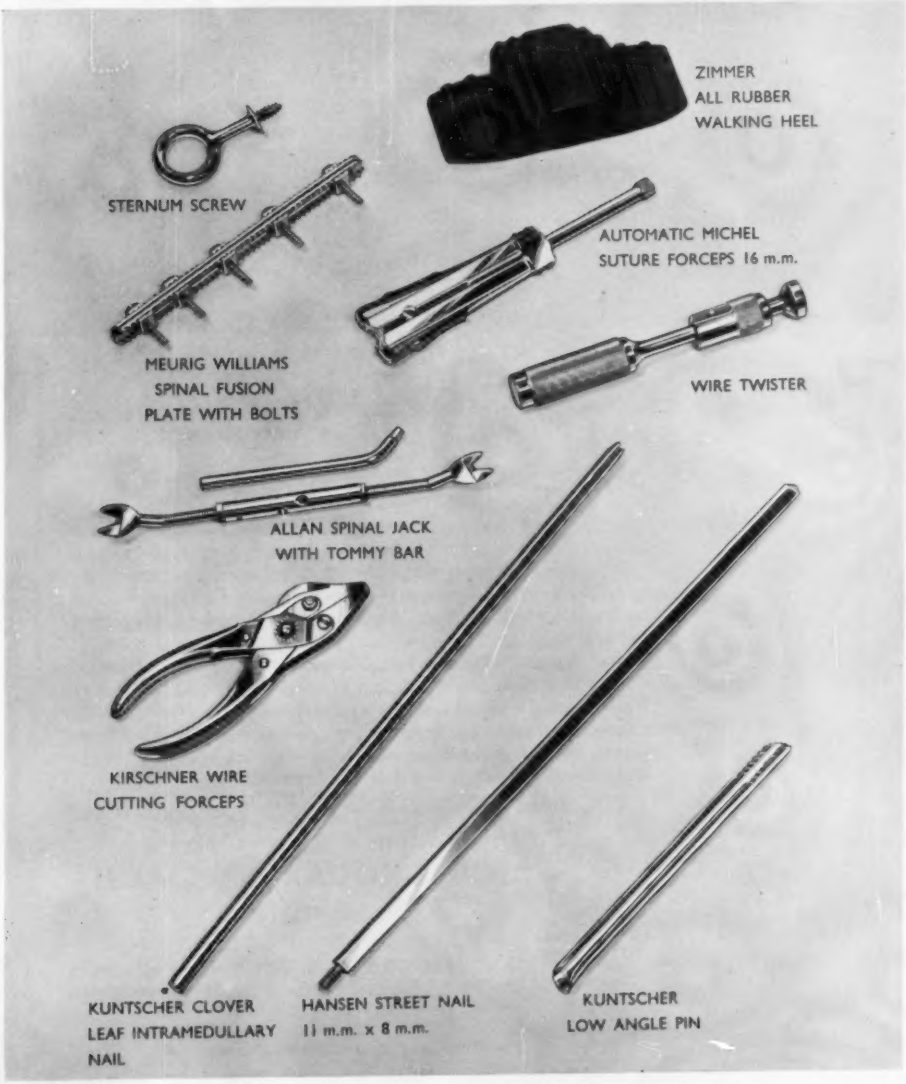

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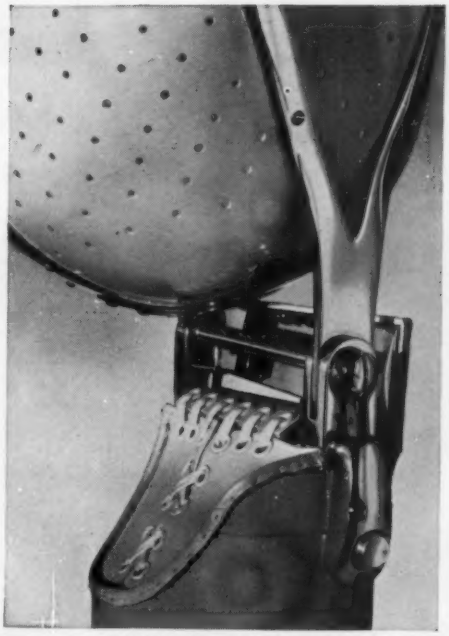

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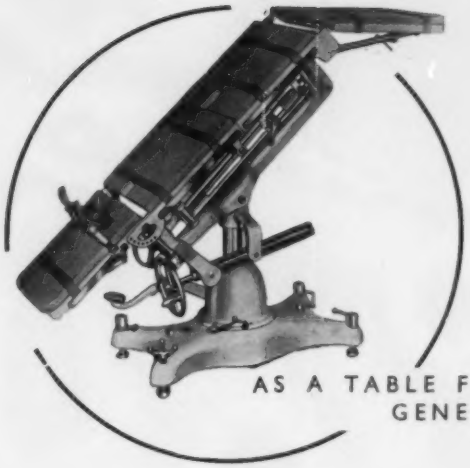

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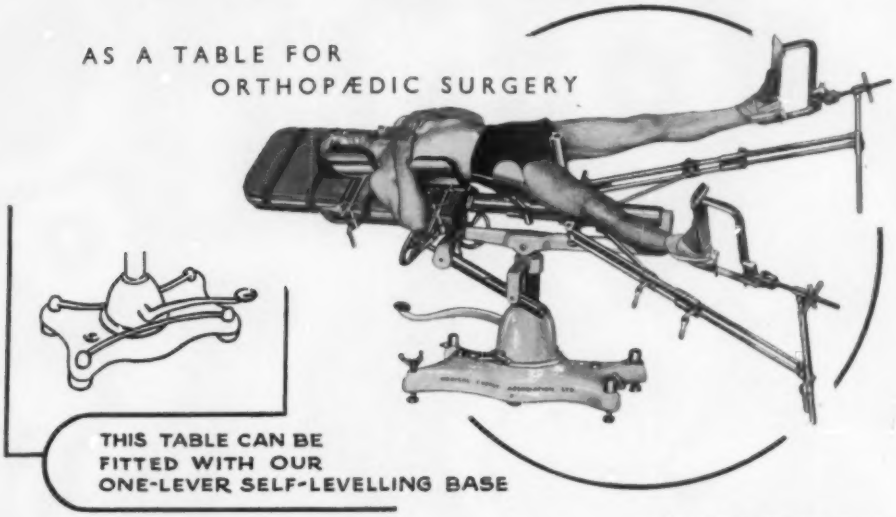

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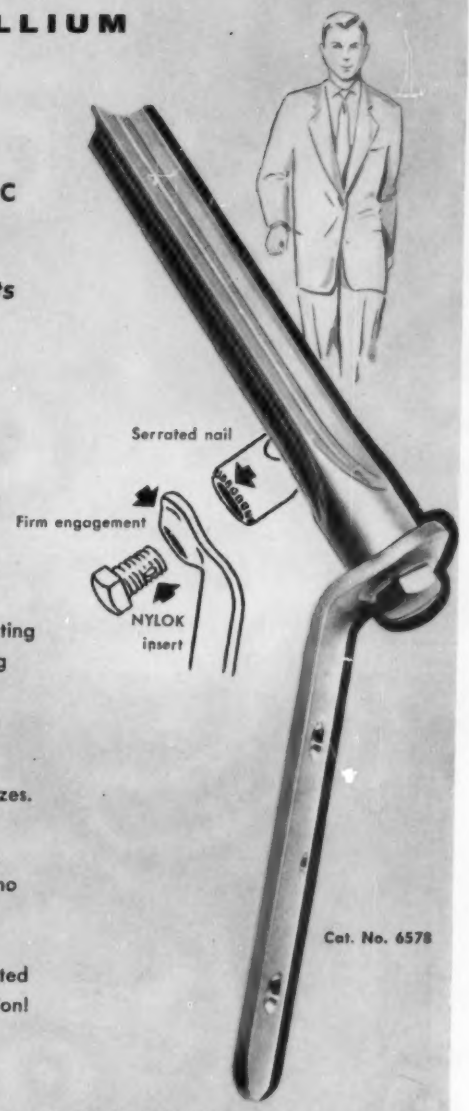

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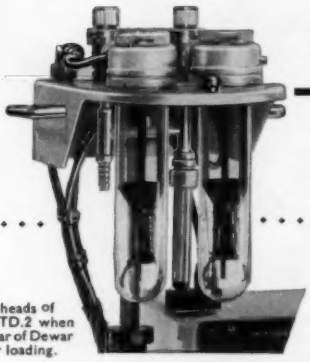

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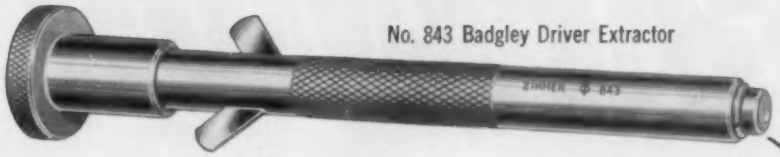

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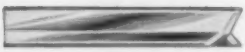

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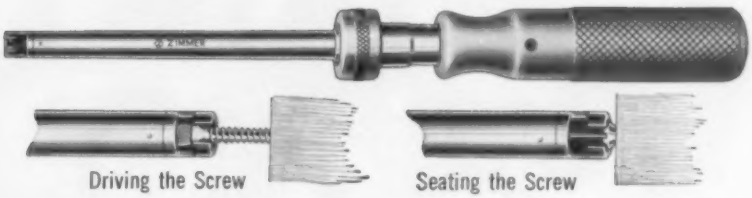

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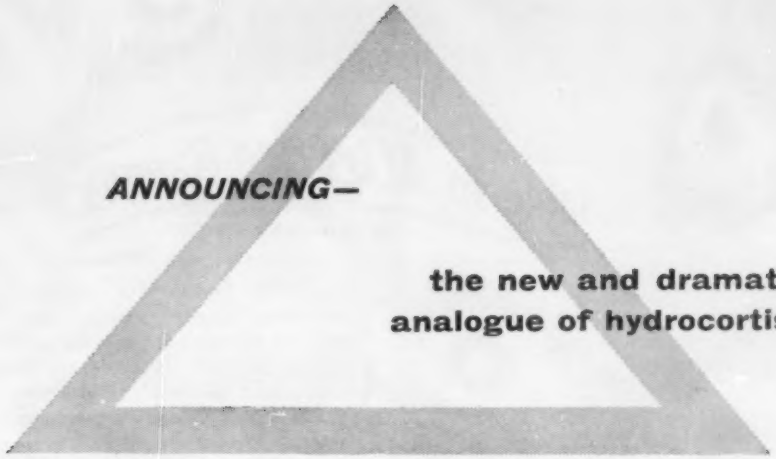

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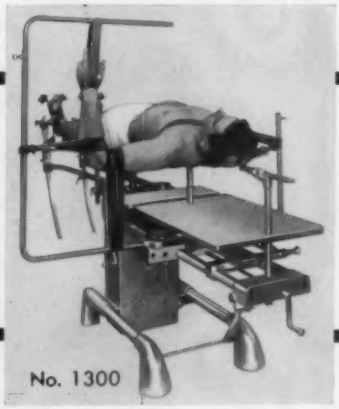

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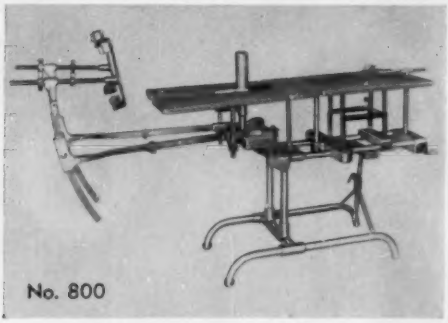

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